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Title: Assessment of Genetic Resistance to Strawbreaker Foot-rot (*Pseudocercosporella herpotrichoides*) in Selected Winter Wheat (*Triticum aestivum L.*) Cultivars.

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Strawbreaker foot-rot is a major limiting factor to cost efficient winter wheat production in the Pacific Northwest. Development of resistant cultivars has been hindered by the lack of adequate levels of genetic resistance and screening techniques which can consistently detect desired genotypes.

Studies were conducted to determine if the reported strawbreaker foot-rot resistance of the cultivar "Rendezvous" is effective on isolates of *Pseudocercosporella herpotrichoides* found in the Pacific Northwest. Protected, naturally infected and artificially inoculated treatments were employed to determine the level of resistance of 10 cultivars including Rendezvous. Different concentrations of inoculum and stages of development were also used to determine if observations on leaf sheath penetration of seedlings obtained in the greenhouse were related to disease severity index readings taken

in the field for selected cultivars. In addition, the nature of inheritance of strawbreaker foot-rot was studied in two crosses involving Rendezvous.

Experiments were conducted at three locations and over two years at one location. Despite cultivar x treatment interaction, consistent levels of infection were observed in all experiments at each location. Significant differences were found for treatments and cultivars for most attributes.

Yield losses, including the components of yield spikes per square meter, 1000 kernel weight, and kernel number per spike were proportional to the severity of the disease. Losses were greater when lodging occurred, which was also associated with disease severity. However, even in the absence of lodging losses were recorded in the naturally and artificially inoculated plots. Traits measured involving Rendezvous and Vpm/Mos 95//*2Hill were only slightly influenced by the treatments.

Under greenhouse conditions, it was possible to distinguish the level of resistance of Rendezvous from susceptible cultivars at concentrations of 100 spores/ml, two weeks after inoculation at the seedling stage. Leaf sheath penetration of seedlings was found to be closely associated with the disease severity index obtained under field conditions.

Generation means analysis performed in crosses involving Rendezvous indicated that additive and additive x additive gene action were responsible for most of the genetic variability associated with resistance. Narrow-sense heritability estimates also confirmed these findings. It would appear that Rendezvous has at least two major genes for resistance to strawbreaker foot-rot.

Assessment of Genetic Resistance to Strawbreaker Foot-Rot (*Pseudocercosporella Herpotrichoides*) in Selected Winter Wheat (*Triticum aestivum L.*) Cultivars

By

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IN DEDICATION TO:

Dolores Rosa Maria, my wife

Patty and Jordi A., my children

Francisco and Magdalena, my parents

Faustino and Esperanza, my wife's parents,

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INTRODUCTION

Strawbreaker foot-rot disease of winter wheat caused by the fungus *Pseudocercosporella herpotrichoides* has been reported to cause severe economic losses in many parts of the world. In the United States strawbreaker foot-rot is a serious problem in the Pacific Northwest winter wheat growing areas. Annual applications of a protective fungicide sprays in winter wheat for disease control are a standard production practice. The development of cultivars with greater tillering capacity, increased used of fertilizers and early planting for soil erosion control have improved grain yields but have promoted the incidence of strawbreaker foot-rot.

During the 1970's benzimidazole fungicides were extensively used against the pathogen in Europe. However in 1981 a lack of control was observed and the frequency of fungicide-resistant isolates increased. Due to this increase of resistance in isolates of strawbreaker foot-rot, the benzimidazole fungicides are not longer effective in controlling the disease. Prochloraz offers an advantage, giving a moderate control of both benzimidazole-resistant and sensitive isolates. The same problem with increased resistance in the pathogen population to fungicides has been noted in eastern Oregon. This change in isolates, the added cost of fungicides and the desire to avoid the use of pesticides lends greater urgency to the development of

resistant cultivars. Unfortunately there are no adapted cultivars to the Pacific Northwest with acceptable levels of resistance to strawbreaker foot-rot.

Development of resistant cultivars is an important objective of the breeding programs in the region. The most economical and environmentally sound method of disease control is through the development of resistant cultivars. Unfortunately information on the nature of inheritance of resistance to strawbreaker foot-rot is limited. Different modes of inheritance to the disease are reported in the literature. These range from simple Mendelian ratios in the wheat line VPM1 to complex quantitative inheritance patterns involving at least four chromosomes in the cultivar "Cappelle-Desprez".

Although resistance has been reported in Cappelle-Desprez, considerable yield losses still occur with this cultivar. Resistance to strawbreaker foot-rot found in *Aegilops ventricosa* in 1936 was successfully transferred to the hexaploid wheat line VPM1 thirty years later. Resistance genes from Cappelle-Desprez and VPM1 have been transferred through hybridization and subsequent selection to the wheat cultivar "Rendezvous". This cultivar was found to be more resistant than either parental source alone when grown in Europe and represents the best source of resistance presently available.

To exploit the resistance found in Rendezvous, it is necessary to determine if the resistance is also effective against the pathotypes found in the Pacific Northwest. More information on the inheritance of resistance to strawbreaker foot-rot and the number of genes involved are necessary to expedite the breeding efforts.

Furthermore, efficient selection for strawbreaker foot-rot resistant genotypes requires development of methods and techniques for both greenhouse and field screening techniques that are reliable and fast both at the seedling and adult plant stages of growth. Knowing the mode of inheritance of resistance in Rendezvous will help in designing suitable breeding strategies for incorporation of strawbreaker foot-rot resistance into high yielding cultivars adapted to the Pacific Northwest wheat production areas.

The objectives of this study were to: 1) determine if strawbreaker foot-rot resistance reported in the cultivar Rendezvous and VPM was effective on isolates found in the Pacific Northwest by assessing losses caused by the disease; 2) assess techniques using different inoculum concentrations to screen for resistance at the seedling stage under controlled environment conditions, and 3) determine the nature of inheritance to strawbreaker foot-rot resistance in two crosses involving Rendezvous.

LITERATURE REVIEW

Incorporation of genetic resistance to strawbreaker foot-rot, a disease caused by *Pseudocercosporella herpotrichoides* (Fron), Deighton, is an important objective of many winter wheat (*Triticum aestivum* L.) breeding programs. Environments where strawbreaker foot rot may be a serious problem are generally characterized as having mild, humid winters, and long, cool, and wet springs. Such areas are found in Europe, North and South America, Australia, and New Zealand (Doussinault, 1970; Doussinault et al. 1983). Bruehl et al. (1968) described strawbreaker foot-rot as the most destructive soil-borne pathogen of winter wheat in the state of Washington. Fungicide is currently applied to 70% of the winter wheat crop in eastern Washington and Northern Idaho to control the disease (Herman and Weise, 1985). In England, losses caused by Strawbreaker foot-rot varied from 0.05 to 2.0% from 1981-86. However, in 1987 the disease was more severe, causing an estimated national yield loss of 2.35%. This yield loss is equivalent to a financial loss of £31.5 million, without considering the cost of sprays applied against the pathogen, or losses caused by lodging and quality of wheat (Fitt et al., 1988).

Wheat is the most susceptible of the small grains to strawbreaker foot-rot barley (*Hordeum vulgare* L.) is somewhat less susceptible, while oats (*Avena sativa* L) and rye (*Secale cereale* L.) are only slightly susceptible to the pathogen (Scott et al., 1975). However, different pathotypes of the fungus have been identified that are able to attack different crops. Pathotypes that mainly infect wheat have been

designated "W", whereas pathotypes infecting rye as well as wheat have been designated "R" (Cunningham, 1981).

Based on colony and spore morphology, Nirenberg (1981) described a new variety, *P. herpotrichoides* var. *acuformis*, and two new species, *P. anguioides* and *P. aestiva*. Fitt et al. (1987) confirmed the observations of Nirenberg (1981) that W-pathotypes, with a larger proportion of curved conidia, corresponded to *P. herpotrichoides* and that R-pathotypes, with predominantly straight conidia, corresponded to *P. herpotrichoides* var. *acuformis*. Nirenberg (1981) and Hollins et al. (1985) described the mycelia of W-type as growing in a rapid, even fashion on agar, whereas R-types grew more slowly in an irregular manner. Fitt et al. (1987) discovered that W-types were generally more pathogenic to wheat compared to R-types, regardless of whether conidia or mycelial inoculum was used. Considerable variation for pathogenicity within isolates of either type was observed, which necessitated the use of large numbers of isolates in tests. Few differences in length, cell number or germination between the conidia of the two pathotypes were found. Dosba and Dossinault (1981) found a higher frequency of R-types infecting wheat lines that had *Aegilops* cytoplasm compared with those that had wheat cytoplasm.

Strawbreaker foot-rot overwinters as a saprophyte on stubble and straw and can remain viable up to three years (Glynne, 1944). It is not efficient in decomposing cellulose and is therefore a poor primary colonizer. However, it may restrict the growth of other soil microflora (Macer, 1961). The fungus survives on stubble in a very inactive form (Deacon, 1973), but Cox and Cock (1962) were able

to recover infected straw from the soil surface 18 months after harvesting a cereal crop. Only a small percentage of the fungus was able to produce spores, but all were sufficient to reestablish infection.

Conidia of strawbreaker foot-rot are freely produced in the autumn and spring and are primarily dispersed by splashing rain. The dispersal range is only 0.9 to 1.2 m, although spores can be wind-borne after mechanical disturbance (Rowe and Powelson, 1973b). Dispersal through the soil is of minor importance (Hollins and Scott, 1980). It was originally assumed that secondary inoculum is produced from primary lesions, but this method of infection is now considered minor (Bruehl et al., 1982b; Hollins and Scott, 1980; Rowe and Powelson, 1973a). Rowe and Powelson (1973b) established point sources of inoculum in the field (infected oat kernels), and at monthly intervals took stem samples from wheat growing at increasing distances from the source. Lesions became visible on the wheat plants four to six weeks after infection. Although these lesions are capable of producing conidia, Bruehl et al. (1982b) found that removal of the primary inoculum in the spring reduced or eliminated the normal infection rate. Secondary tillers could be infected by primary inoculum or by direct contact with infected tissue. The low level of production of secondary inoculum indicated that strawbreaker is a simple interest disease and conditions favorable for infection occurred sporadically in October, November and from late February to mid-May in the Pacific Northwest (Rowe and Powelson, 1973a; Bruehl et al. 1982b).

McCoy and Powelson (1973) discovered that with soil-borne inoculum, more spores of strawbreaker foot-rot were required to establish infection in a crop grown on a silt loam than on sand or sandy loam soil. They concluded that the decrease in disease incidence in the silt loam soil was correlated with higher rates of microbial immobilization of sucrose, resulting in reduction of the rhizosphere influence.

Lesions of strawbreaker foot rot usually develop near the soil surface on wheat culms and are found rarely on leaves. Tissue may show differential resistance within the host, and environmental factors such as light intensity, temperature, and relative humidity, may be involved in affecting host microclimate and subsequent infections (Higgins, 1984).

Epidemiology of Strawbreaker Foot-Rot

Investigations on the epidemiology of strawbreaker foot-rot were conducted by Schrodter and Fehrmann (1971a, 1971b). They reported that temperature was the most limiting factor in an infection given adequate supply of spores. Maximum spore production occurred at 3 to 4 °C, while infection occurred over a temperature range of 4 to 13 °C with an optimum at 8 to 9 °C. Infection was independent of the intensity of rainfall as long as there was a relative humidity of 80% for at least 15 hours. Airflow, by dynamic and thermal turbulence, accounted for most of the distribution of conidia (Schrodter and Fehrmann, 1971a).

The fungus sporulated over a temperature range of 10 to 15 °C with an optimum of 10 °C (Rowe and Powelson, 1973a; Scott, 1971). Rowe and Powelson (1973a) developed a daily thermal sporulation coefficient (DTSC) as a function of the total number of hours of favorable and unfavorable temperatures that occur daily. The fungus had moderate sporulation between 8 and 12 °C if the temperature did not go below 0 °C for more than 14 hours or above 20 °C for more than 10 hours. The relative humidity near the soil had to be near saturation.

The optimum temperature for spore germination and mycelial growth was found by Bruehl and Manandhar (1972) and Scott (1971) to be around 20 °C, but mycelial growth had a lower requirement for moisture that did spore gemination (Higgins, 1984). Maximum mycelial growth occurred at a water potential of -1 to -10 bars, which correlated with the water potential of wheat leaves under no water stress; but growth still occurred at -90 bars, which has been previously thought to be unlikely. Growth was favored by decreased water potential as the temperature increased (Bruehl and Manandhar, 1972).

Higgins and Fitt (1985a) showed that although the optimal temperature for fungal growth in culture was 20 °C, cooler temperatures were more favorable for infection in the field. Even though the penetration rate of mycelia increased with slightly warmer temperatures, leaf sheaths tended to die and slough-off more quickly so that the period when infected sheaths were in contact with developing stems was reduced. Higgins et al. (1986) reported that the stage when stems and leaf sheaths were in contact was essential for disease establishment, and was also reduced under

low seeding rates that allowed for greater aeration within the crop and enhanced desiccation of the basal leaf sheaths. They also concluded that early fall planting increased lesion development in the sheaths and hastened stem elongation, which allowed for a longer time period for infection of the stem. The potential for yield loss was therefore increased.

Spores attach to the plant by a mucilaginous substance and germinate on the surface of coleoptiles or leaf sheaths. Penetration of the epidermis is by mechanical and enzymatic action, and successive leaf sheaths are invaded from the stromas formed between them (Doussinault and Dosba, 1977). Guillot-Salomon and Doussinault (1981) reported that penetration of the plant epidermis was due primarily to enzymatic degradation, rather than mechanical degradation, of the host cell wall. The pathogen grows slowly, reaching the stem just after the beginning of its elongation stage and causes tissue disorganization and consequently lodging (Doussinault and Dosba, 1977; Murray and Bruehl, 1986). Infection by strawbreaker foot-rot may reduce kernel weight (Murray and Bruehl, 1986; Magnus and Hansen, 1973) and kernel number (Dosba and Doussinault, 1981). However, Doussinault (1973) did not observed a reduction in kernel weight in his studies. The lesions may cause white or prematurely ripened heads. Yield loss in wheat infected by strawbreaker foot rot is due primarily to reduction in grain size and weight (Dosba and Doussinault, 1973; Magnus and Hansen, 1973).

Doussinault (1973) identified three main factors that affect host plant response: a) the probability of the plant being infected, b) resistance of the leaf

sheaths to infection, and c) resistance of the stem to attack. Murray and Bruehl (1983), maintained that resistant cultivars should have open crowns, sparse tillering, coarse straw with tough leaf sheaths, broad leaves, short stature, late maturity, and anthocyanin pigmentation of the stems. They also reported that hypodermis width, the earliness of thickening and lignification, and the number of hypodermis cell layers were correlated with disease indices.

Three stages of lesion development following infection were described by Fitt (1985): a) successive leaf sheath penetration, b) stem lesion establishment, and c) stem lesion development. The first stage lasted several months and was not influenced greatly by small changes in temperature and relative humidity. The second stage occurred soon after stem elongation. It was probably the most important stage in determining final yield loss, because this was when the developed stem was in contact with basal leaf sheaths prior to their sloughing-off. The third stage lasted until harvest and was dependent more on accumulated temperature than on time or rainfall.

Four types of loss from strawbreaker were described by Jorgensen (1964); a) loss from severe early attack that killed the shoots before elongation, b) loss from severe early attack that killed the shoots after elongation but before heading was complete, c) loss due to the formation of necrotic spots at the base of the stem, and d) loss due to lodging caused by severe stem attack. He noted a crop could compensate for the yield loss due to early attacks, so disease incidence in the spring was not a reliable indicator of yield loss. Yield loss due to lodging was considered

an indirect effect of the fungus, whereas yield loss due to damage caused by the lesions was a direct effect (Scott and Hollins, 1974).

Murray and Bruehl (1986), reported that as disease severity (the mean lesion score for 50 to 100 individually rated tillers per plot) increased so did the frequency of tillers that had severe lesions. But even when the overall disease index was high, some tillers remained relatively healthy. Conversely, some tillers were badly affected even when the rest of the cultivars showed little disease. Lines such as VPM, which are prone to lodging in the absence of disease, could cause significant cultivar x inoculation interactions. They concluded that disease index was more reliable than yield for identifying resistant types. In all trials the disease reduced stand density and kernel size, especially in highly susceptible cultivars. Generally, the yield of all cultivars was most affected under severe disease conditions, though more resistant cultivars incurred smaller reductions in stand density and kernel weight. Under less severe disease conditions, disease x cultivar interactions did not occur and lower yields were due primarily to lower plant densities. Yield was affected by both host resistance and yield potential as susceptible cultivars with high yield potential could outyield resistant cultivars.

Control of Strawbreaker Foot-Rot

Cultural practices and development of cultivars with a greater tillering capacity are major factors affecting the incidence of strawbreaker foot rot in the

Pacific Northwest. This has extended the geographic range of the pathogen from climates with moist winters to those with the more marginal rainfall of 250 mm per year (Bruehl et al., 1968). Increased use of fertilizers and early seeding for soil erosion control have improved yields but promoted strawbreaker foot rot. Winter wheat grown in an annual cropping system is less at risk to infection because seeding dates are later than that in the wheat-fallow system practiced in the dryland areas. Rotation with spring cereals or peas (*Pisum sativum* L.) is common in higher rainfall areas of eastern Oregon and Washington; both rotations reduce disease incidence; however it is not generally economic to remove the fields from winter cereals for long periods of time (Bruehl et al., 1986). Bockmann and Mielke (1983) maintained that a two year break from winter wheat was required to help control strawbreaker foot-rot in Germany. It has also been observed that cereals planted in rotation generally did not reduce the incidence of strawbreaker in winter wheat (Vechet, 1983).

Although the effects of reduced tillage on strawbreaker foot-rot development were originally unclear, Herman and Wiese (1985) found that reduced and no-till management resulted in successively lower disease levels when compared to conventional tillage that left less than 30% of the crop residue on the soil surface. They stated that straw on the soil surface created a barrier between the plant and spores in the soil. Grain yield was lower under no-till conditions, but input costs were also reduced.

Early planting in September increased the incidence of strawbreaker foot rot because the plants were larger and had more tillers during the time of maximum spore production (Bruehl et al., 1968; Rowe and Powelson, 1973b). Plants seeded later were more likely to escape infection but had a lower yield potential and the opportunities for soil erosion increased (Herman and Wiese, 1985).

The use of fungicides to control strawbreaker foot-rot can be economic (Herman and Wiese, 1985), but it has been stressed that there is no advantage to using a fungicide unless strawbreaker foot-rot is actually a problem and at least 10% (Scott and Hollins, 1978) to 20% (Higgins et al., 1986; Huber and Mulanax, 1972) of plants are infected at the beginning of the stem elongation stage. Martin (1986) cautioned that yield reductions may occur when fungicides are applied when disease incidence is low. The systemic fungicide benomyl, a carbendazim type with the active ingredient methyl-2-benzimidazole-carbamate, has generally been proven to be the most efficacious against strawbreaker foot-rot (Born and Powelson, 1985; Bruehl et al., 1982a; Huber and Mulanax, 1972; Murray, 1986).

Fehrmann and Schrodter (1872) obtained an additive effect when they used benomyl in conjunction with chloromequat (CCC = chlorocholinechchloride) and especially when applied after a high rate of nitrogenous fertilizer (120 Kg N/ha). The systemic fungicide was most effective when applied right after a period when there was a high probability of infection. Disease did not have to be eliminated completely to obtain an optimum economic yield. Witchalls and Close (1971) concluded that benomyl was more effective than CCC because the latter shortened

and stiffened the wheat stems in order to reduce lodging. In Britain, Barnes et al. (1983) found that the use of prochloraz in combination with carbendazim had advantages over the use of the fungicides individually.

Bruehl et al. (1982a) found benomyl to be effective over a wide range of applications dates, which made it more useful than thiabendazole (TBZ). Applications in March and April were best, whereas applications in November, December. and May were ineffective. According to Powelson and Rohde (1972), an application in February was able to reduce culm infection from 14 to 95% and increase yield from 2.96 to 5.51 t/ha in early seeded wheat. Bruehl and Cunfer (1972) reported that late winter spraying with benomyl at the rate of 0.14 kg/ha a.i. was the most economic treatment. Benomyl would be translocated down in the stems, and because the fungus was able to survive in the straw for up to 3 years, spraying could be beneficial for more than 1 year. Janicke et al. (1984) found that carbendazim was most effective when applied to leaf sheaths or into the axil of the lowest leaf, but there was no relationship between disease control and the amount of fungicide applied.

A serious development has been the increasing resistance of strawbreaker foot-rot isolates to carbendazim fungicides. The phenomenon was first reported in Germany in 1977, followed by a sudden increase in resistance to the fungicides in 1982 (Fehrmann, 1985). Tolerant isolates have been found in England and Wales (King and Griffin, 1985), the Netherlands (Sanders et at., 1986), France (Cavelier et al., 1985), and in Washington (Bruehl et al., 1985). In general, resistance increased

with a accumulation of fungicide spraying per year and over years, (Fehrman, 1985; Bateman et al.,1985).

Tests for fungicide resistance have been combined with work on the pathogenicity of strawbreaker foot-rot isolates. Higgins and Fitt (1985b) conducted pathogenicity tests on seedling and adult plants using isolates that were both sensitive or resistant to carbendazim. Spores were better than mycelial inoculum as lesion development was affected by the number of spores reaching the crop i.e. the threshold level. Seedling pathogenicity was not clearly related to pathogenicity on adult plants, and fungicide resistant isolates were found to be both more and less pathogenic than those susceptible to the fungicide. Bateman et al. (1985) found that resistant and sensitive isolates to carbendazim were equally pathogenic on wheat, and both types were sensitive to prochloraz fungicide.

Sanders et al. (1986) confirmed that benzimidazole resistant types were more common among R type isolates. They also found that EBI (ergosterol biosynthesis inhibitor) fungicides were less effective on benzimidazole resistant R types than on sensitive R or W types. Therefore, both EBI and benzimidazole fungicides may provide R types isolates with a selective advantage.

Hoare et al. (1986) conducted studies on land with no history of cereal production or carbendazim application. They applied inoculum containing equal amounts of R- and W-type spores to winter wheat. Repeated treatments of different combinations or separate applications of carbendazim and prochloraz fungicides were carried out for three years. Resistant isolates from plots treated only with

carbendazim quickly increased. When a mixture of carbendazim and prochloraz was used, resistant isolates increased more slowly and after three years were at a similar level to carbendazim alone. With prochloraz only, or in plots without fungicide, isolates resistant to carbendazim occurred consistently at a frequency of $\leq$ 14%. No isolates were found to be resistant to prochloraz. The proportion of W-type isolates remained high in untreated plots, but decreased with the application of the fungicides. Both fungicides caused an increase in R-type isolates, but more isolates were carbendazim resistant if they had been treated with carbendazim rather than with prochloraz. From these results Hoare et al. (1986) suggested that R-type and carbendazim resistance responded independently to selection and that use of prochloraz would increase R-type isolates, but would not necessarily increase isolates with carbendazim resistance. This was contrary to the findings of Hollins et al. (1985) who had suggested that carbendazim resistance and R-type might be controlled by genes that are associated or linked.

Sources of Resistance to Strawbreaker Foot-Rot

"Cappelle-Desprez" (Cappelle) was regarded as the most resistant cultivar to strawbreaker foot-rot in the 1970's, but its resistance level was still insufficient (Doussinault, 1970). Sprague (1936) found that most related wild grasses were less susceptible than wheat to strawbreaker foot rot, with the exception of *Agropyron* species.

Aegilops ventricosa has been tested specifically for resistance to strawbreaker foot-rot and has proven to be the most resistant of several species of *Aegilops* and *Triticum*. These species were resistant or slightly susceptible to the pathogen in both seedling and adult stages (Groll et al. 1985; Khan and Bouriquet, 1984).

The genus *Aegilops* has also been shown to have genes conferring resistance to other diseases. Bochev et al. (1982) found resistance to common races of powdery mildew (*Erysiphe graminis* f. sp. *tritici*), leaf rust (*Puccinia recondita* f. sp. *tritici*), and stem rust (*Puccinia graminis* f. sp. *tritici*) in 22 species of Aegilops.

Dosba and Doussinault (1973) assessed 43 lines of five *Aegilops* species and found the line "Vent 11" to be the most resistant to strawbreaker foot-rot. They concluded that the D genome of *Aegilops ventricosa* was different from that of *Aegilops squarrosa*, which is considered to be the donor of D genome in wheat. They also noted that since 1936 no strawbreaker foot-rot isolates have shown to be virulent to *Aegilops ventricosa* in Britain, France, Belgium or in the United States. Maia (1967) described the different steps involved in the incorporation of *Aegilops* germplasm into wheat. *Aegilops ventricosa/T. timopheevi//* and 'Thatcher' wheat were crossed to obtain plants that had 42 chromosomes and were resistant to strawbreaker foot rot. In 1953 a cross was made between *Ae. ventricosa* (DDM^vM^v) and *T. persicum* and then crossed to *T. dicoccum* used as the male parent. Sterility of the F_1 in this amphiploid was overcome by doubling the chromosome number, then backcrossing three times to 'Marne' wheat followed by six generations of selfing. The M^v chromosomes of *Ae. ventricosa* were eliminated with the introgression of

genes for strawbreaker resistance into hexaploid wheat. The line "VPM" that was obtained has strawbreaker resistance, indicating that resistance genes were located in the D genome. However, due to the late maturity of "VPM" its potential is as a parent more than for direct commercial use (Maia, 1967).

Kimber (1967) incorporated strawbreaker resistance of *Ae. ventricosa* into hexaploid wheat using *Triticum turgidum* (A and B genome) as a bridge species. Viable and fertile amphiploids were obtained, but a major obstacle was the lack of homology among wheat and alien chromosomes. The resulting level of resistance was better than that of Cappelle-Desprez but less than that of *Ae. ventricosa*, as the lines obtained had been backcrossed to a susceptible type. Resistant plants did not have an additional chromosome, so the genes transferred were not on the M^v genome of *Ae. ventricosa*. Additional resistance gene or genes could be on the M^v genome and would be difficult to incorporate into the hexaploid wheat genome.

Several methods of introgression were used by Rober-Merien and Doussinault (1983): a) crossing *Ae. ventricosa* with tetraploid *T. dicoccum* and *T. turgidum*, b) making addition lines by incorporating the M genome of *Ae. ventricosa* into wheat, c) making direct crosses between *Ae. ventricosa* and monosomic 5B lines, and d) making reciprocal crosses between Cappelle-Desprez and VPM. They found several lines that were more resistant than VPM to strawbreaker, especially among the crosses with Cappelle-Desprez. Complete dominance for resistance was expressed in lines possessing *Ae. ventricosa* cytoplasm. However, when a quantitative measure was used (number of leaf sheaths attacked), partial dominance was observed.

Dosba and Cauderon (1972) manage to make direct crosses between wheat and *Ae. ventricosa*, but subsequent meiotic analysis of the plants obtained raised questions as to the extent of homology between the two species. Dosba et al. (1978) attempted to incorporate the resistance on M^v chromosomes into wheat by making addition lines in 'Moisson'. Lines extracted on wheat cytoplasm were cytologically more stable, but *Aegilops* cytoplasm appeared to promote strawbreaker resistance. Dosba et al. (1980) made an addition line of *Ae. ventricosa* wheat on *Aegilops* cytoplasm. The transmission rate of the additional chromosome was 0.40 in female gametes and 0.16 in male gametes. The addition lines differed in flag leaf area and content of chlorophyll a and b, so it was assumed these characters were linked to the added chromosomes.

Inheritance Resistance to Strawbreaker Foot-Rot

There are limited reports on the inheritance and mode of action of strawbreaker foot rot resistant genes in *Ae. ventricosa*-derived lines. Dosba and Doussinault (1978) attempted to combine resistance to strawbreaker foot rot and useful agronomic characteristics. They crossed *Ae. ventricosa* with tetraploid wheat to obtain amphiploids that were crossed twice with hexaploid wheat, selfed, and crossed to semidwarf wheat. Amphiploids had a level of strawbreaker foot rot resistance greater than that of VPM and similar to that of *Ae. ventricosa*. The chromosome number of the amphiploids stabilized at 42 after five generations of

selfing. Some of the lines were resistant to powdery mildew, leaf or stripe rust, and had high protein content.

Dosba (1982) found considerable meiotic instability in M^v addition lines of *Ae. ventricosa* extracted on *Ae. ventricosa* cytoplasm. Many univalents and multivalents were evident in early generations and stability at the 44 chromosome level varied among the lines. Cytological analysis revealed a high synapsis rate, higher transmission rate of female than male gametes, and low meiotic stability in some lines.

Monosomic analysis has indicated that chromosomes 2B, 5D and 7D are involved in resistance to strawbreaker foot-rot in Capelle-Dessprez. Dominant gene action favored resistance and was nearly complete. The study was conducted using F_2 seedlings derived from the monosomic cross and the resistance score was obtained from the number of leaves penetrated by the fungus (Law et al., 1976).

Monosomic analysis of the cultivar 'Roazon' (derived from VPM/Moisson) implicated chromosomes 7A, 7D and 5D in carrying resistance genes, but the actual number of chromosomes involved could not be concluded because the F_2's were more susceptible in the monosomic state. Resistance scores were determined in the seedling and adult stages from the number of leaf sheaths and stems, respectively, that were penetrated by the fungus (Jahier et al., 1978).

Delibes et al. (1977a) used biochemical markers and concluded that the gene transfer from the M^v genome of *Ae. ventricosa* to hexaploid wheat occurred by chromosome substitution and also through recombination. However, in another

report, Delibes et al. (1977a) indicated that resistant allele was carried on the D genome and that inheritance was simple, possibly due to one major gene. In their study, seedling resistance score was also obtained from the number of leaf sheaths that were attacked, with adult plant resistance determined by cross sectioning stems and dividing them into two classes based on the number of tillers that were more or less than 50% infected.

Using a diallel analysis of reciprocal crosses among four cultivars that differed as to the extent which strawbreaker foot-rot penetrated the seedling leaf sheaths, Saragoussi (1986), concluded that *Ae. ventricosa* contributed a dominant gene for resistance to strawbreaker foot-rot. Lines that did not contain *Aegilops* genes had an additive system of resistance.

Hollins and Scott (1986) also concluded that strawbreaker resistance was located on chromosome 7D and was controlled by a single dominant gene. However, Murray (1983) maintained that those who reported simple inheritance had crossed resistant with resistant lines, which did not allow for the full range of segregation and that the inheritance of resistance was probably not simple.

Doussinault et al. (1983) rated *Aegilops*-derived lines in the seedling and adult stages, using the same method as Delibes et al (1977a), and found cytoplasmic effects in the F_1, F_2 and backcrosses generations. They maintained that the cytoplasmic background and method of scoring resistance affected the apparent expression and interpretation of the number of resistant genes.

The stability of the resistance of Cappelle and the apparent absence of physiologic races of the fungus indicates that the pathogen is race nonspecific and that there is no reason to doubt the durability of the resistance of wheat cultivars (Scott and Hollins, 1977). However, Scott et al., (1976) reported that certain lines of *Ae. squarrosa* were susceptible to 'R' pathotypes of the fungus, so resistance introduced into wheat from these lines could break down. They hypothesized further that the strawbreaker foot-rot fungus may lack adaptation to the type of resistance shown, or that such adaptation has no selective advantage, or that adapted forms occur at too low frequency to be recognized. Pathogenic adaptation within necrotrophic species of cereal pathogens is expressed more commonly at the level of host genus or species than at the cultivar level, so it is uncertain whether strawbreaker foot-rot resistance from alien genera will be as durable as resistance already within the species. The limited dispersal of the fungus should restrict erosion of resistance, especially if adapted forms of the pathogen do not have a great selective advantage.

Various methods have been used to improve the levels of resistance to strawbreaker foot-rot in wheat cultivars. Magnus and Hansen (1973) emphasized that selection for tolerance should include a number of factors: winter kill, disease rating, percent lodging, grain size, and seed yield.

Murray (1983) selected material on the basis of hypodermis width and disease index. Since there were environmental variances for these traits he emphasized the need for progeny testing in different years and locations.

Based on the knowledge that strawbreaker foot-rot reduces grain size and density, Ecochard and Mansat (1958) used a mass selection technique to identify disease resistant progeny in a susceptible/resistant cross. F_2 seeds were divided according to the size. The population was advanced for two years and the seed was sorted by density in an air column. Populations were grown out in the field the third season and lines identified as resistant to strawbreaker foot-rot had grain development and yield superior to the most resistant parent. Base on their results, they concluded that mass selection could be employed to enhance resistance to strawbreaker foot-rot if: a) variability for resistance was available in the parental lines, b) disease pressure was applied each season to plots of at least 80 - 100 m^2, c) selection was applied for grain with high density, and d) selection was conducted for several years. However, using natural and mass selection techniques Roberts and Allan (1990), concluded that these methods did not enhance resistance to strawbreaker foot-rot after two cycles of selections for seed size. They noted that these selection methods favored non-semidwarf and plants that were usually susceptible to the pathogen.

Doussinault and Douarie (1978) developed a diallel cross among eight wheat lines of diverse origins. VPM (*Aegilops ventricosa* x *Triticum persicum* x *T. aestivum* cv. Marne) and Cappelle-Desprez were include. Lesion score was determined (on adult plants) by dividing F_2 stems into those with above and below 50% lesion coverage. VPM and Cappelle-Desprez were the more resistant cultivars at both seedling and adult stages, and crosses between them were better than VPM per se.

Both seedling and adult types of resistance were expressed. They reported large differences in general combining ability among cultivars. No differences between reciprocal crosses were noted, and crosses involving VPM sometimes gave more resistant progeny with *Aegilops* cytoplasm.

Scoring for disease resistance at the whole plant level is tedious, time consuming, and not always reliable. There is a need for a more effective selection technique that is faster than traditional methods of growing wheat plants to maturity and assessing the stubble for disease severity. As suggested by Moore and Collins (1983) genetic markers i.e. isozymes that are closely linked with genes for resistance offers several advantages over conventional methods. These include: a) inheritance is usually codominant without epistasis or pleiotropy, b) they can be assayed at any growth stage and in any tissue, c) usually, this method is not destructive, and d) identification and selection of plants carrying functional segments of alien chromosomes can be practiced. Growing plants for a full season can be avoided because isozymes assays are performed at the seedling stage.

One approach could be selecting for endopeptidase activity. In VPM-derived wheat lines, strawbreaker foot-rot resistant genes were located on chromosome 7D (Jahier et al., 1978). Loci for endopeptidase were located on chromosome 7BL and on long arms of 7A and 7D (McMillin and Allan, 1987) and were shown to be closely linked to strawbreaker foot-rot resistance in certain wheat lines (McMillin et al., 1986).

MATERIALS AND METHODS

The materials and methods section is divided into three studies. I. Effect of strawbreaker foot-rot on grain yield and its components on selected winter wheat cultivars grown under field conditions. II. Pathogenicity of strawbreaker foot-rot to wheat seedlings including the effect of inoculum concentration and disease progress over time. III. Inheritance and nature of gene action associated with the resistant reaction to strawbreaker foot-rot in crosses with the cultivar Rendezvous.

Study I. Disease Loss Assessment

To determine the effect of strawbreaker foot-rot on grain yield and the components of yield, 10 winter wheat genotypes were selected. Descriptions and pedigrees for the experimental material are presented in Appendix Table 1. The study was carried out over two consecutive seasons. In 1987-88 the experiment was conducted at the Columbia Basin Research Center (CBRC), near Pendleton OR. During 1988-89, the investigations were conducted both at the Hyslop Crop Science Laboratory (HCSL), near Corvallis OR, and at the CBRC. Soil types at the experimental sites are a fine, silty mixed mesic Aquultic Argixeroll and a coarse silty typic Haploxeroll, respectively. The experiments were planted at both sites in a summer fallow land in 1987-88 and 1988-89. In 1988-89 at CBRC the experiment was planted in a land previously occupied with edible peas.

One hundred Kg ha^{-1} of nitrogen (anhydrous ammonia) and sulphur at the rate of 20 kg ha^{-1} were applied before planting at the CBRC site. During the spring a second application of nitrogen (solution 32) was broadcasted at the rate of 20 kg ha^{-1}. Weeds were controlled using Bromoxynil at a rate of 1.4 l a.i. ha^{-1}.

Prior to planting, 40 Kg ha^{-1} of N and 6 Kg ha^{-1} of ammonium sulphate were applied at HCSL site. Also a total of 120 Kg N ha^{-1} and 24 Kg of S ha^{-1} were applied in the form of 30-0-0-6 fertilizer in one application made during the jointing stage of growth. Weeds were controlled with a fall application of 1.68 kg a.i. ha^{-1} of Diuron at this site.

Each experimental unit in 1987-88 consisted of a four-row plots, 5 m long, with 30 cm between rows. Six rows 5 m long at a 30 cm row spacing were evaluated in 1988-89. A factorial set of treatments were arranged in a randomized complete block design with four replications was employed during both years. In the 1987-88 and 1988-89 seasons the experiments at each location were planted at a seeding rate of 100 Kg/ha. The seeding dates were on September 9 and October 6, respectively at CBRC site, and September 26 at the HCSL location.

Treatments consisted of plots that were fungicide-protected, naturally infected, and artificially inoculated with the pathogen. In 1987-88 season, protected plots were sprayed with benomyl (454 g a.i. ha^{-1}) in the spring. During the second year, plots were protected with a mixture of benomyl (340.5 g a.i. ha^{-1}) and prochloraz (227 g a.i. ha^{-1}). Natural inoculations were accomplished with strawbreaker foot-rot inoculum released by infected host tissues and debris present in the soil. Plots

assigned to be artificially inoculated were sprayed with a liquid suspension containing a mixture of *P. herpotrichoides* spores (PH 85-9-13 an PH 85-13-2) which were obtained according to specifications described by Bruehl and Machtmes (1985). The spore suspension was sprayed using a self propelled back pack sprayer in sufficient amounts to completely cover the plants.

The control of *Septoria spp.* pathogens at the HCSL location was achieved by spraying the whole experiment four times with Propiconazole at a rate of 0.23 Kg a.i. ha^{-1}. Meteroelogical data for both seasons are presented in Appendix Tables 2 and 3.

The following traits were measured on a per plot basis during both growing seasons:

Grain yield per plot: In 1987-88 season, grain yield was determined by hand-sickling two center rows from each plot, bundling the stems and threshing with a Vogel stationary thresher. Entire plots were harvested with a Wintersteiger plot combine in 1988-89.

Number of seeds per spike: Twenty spikes were collected from each plot and threshed using a head thresher. Seeds were counted using an electronic seed counter.

Spikes per square meter: This trait was determined indirectly from the following: weight of twenty spikes, yield per plot, and area harvested. The formula used was as follows: Spikes/m^2 = [(20 x yield per plot/spike weight)/area m^2].

1000 kernel weight: kernels were randomly selected from cleaned samples of harvested grain counted and weighted.

Test weight: Test weight was measured in kilograms per hectoliter (Kg/hl) from the bulk seed harvested.

Disease severity: Individual tillers were assessed for lesion severity based on a scale o 0-4 (Murray and Bruehl, 1983), where 0 = no lesions and 4 = severe lesion girdling the tillers. Prior to harvest a random sample of 50-100 tillers per plot were collected and used to score for disease severity. A disease severity index was calculated for each plot by multiplying the number of tillers in each class by the class number, summing all classes and dividing by the total amount of tillers rated.

Study II. Seedling Evaluation

Effect of inoculum concentration and disease progress over time on wheat seedlings constituted Study II. These experiments were conducted during the spring of 1990 under greenhouse conditions on the Oregon state University campus. Experimental material consisted of two strawbreaker foot-rot susceptible cultivars (Stephens and McDermid), and one resistant cultivars (Rendezvous). Each experimental unit consisted of 10 plants of each cultivar planted in a 15 cm pot containing a sterilized silt loam growing medium. A completely randomized design with six replications was employed. After germination, seedlings were placed in an

inoculation chamber. The greenhouse temperature was maintained at 20 °C during the day and 15 °C at night with a 9 hour daylength. Plants were fertilized regularly with Peters fertilizer 20-20-20 during the course of the experiment.

A virulent isolate (PH 85-9-13 W type) of strawbreaker foot-rot was maintained as mycelial culture on Potato Dextrose Agar (PDA) media, and used to produce spores that served as the inoculum source. Conidial inoculum was produced by using a modification of the method of Reinecke and Fokkema (1979). Small agar discs measuring 10 mm in diameter from 3-4 week old mycelial colonies were cut and placed at the center of a Petri dish containing only distilled water agar (WA). The petri dishes (WA) were then exposed to near ultraviolet light at 15 °C to induce sporulation, which occurred within three days. Aliquots of 1 ml conidial suspension prepared from the water agar plates were spread over PDA media in plastic Petri dishes containing tetracycline hydrochloride to avoid bacterial growth. The conidia germinated and immediately produced new conidia. Plates showed a cream color appearance after two or more days which was the result of formation of masses of conidia. After three days, abundant conidia were obtained from every Petri dish. To obtain good sporulation on PDA media, continuous exposure to high intensity radiation was necessary.

Conidial suspensions were prepared by rubbing the culture surface to free the conidia in sterile water. Conidia from the cultures were filtered through three layers of cheese cloth to prevent contamination by mycelial fragments. Spores were counted with a hemacytometer and final concentration was adjusted to 1,000,000

conidia/ml and serial dilutions were prepared from this final concentration. The solution also contained one drop of Tween 20 as a wetting agent.

Seedlings at the two leaf stage were inoculated with concentrations of 10, 10^2, 10^3, 10^4, 10^5, $5x10^5$ and 10^6 conidia/ml by spraying with the suspensions at the rate of 5 ml per pot. The inoculated plants were incubated in a dew chamber for three days at 18 °C and 9 hours daylength. After the incubation period plants were placed in the greenhouse at 18 °C and 85% relative humidity. Twelve weeks after inoculation, pathogenicity of strawbreaker foot-rot was assessed based on the number of leaf sheaths penetrated by the fungus.

Single regression analysis was used to estimate the relationship between disease severity as measured by the number of leaf sheaths infected and inoculum concentration. A comparison of regression slopes among cultivars was performed by using a t test of homogeneity of regression coefficients tests (Gomez and Gomez, 1984).

To determine the disease development over time, seeds (six per pot) of McDermid and Rendezvous were planted 1 cm deep in a 15 cm diameter plastic pots containing a sterilized silt loam growing medium. The pots were arranged in a completely randomized design and replicated 10 times.

One week old seedlings were inoculated with a mixture of two virulent isolates (W types) of the pathogen (PH 85-9-13 and PH 85-13-2) by spraying with a conidial suspension at the rate of 3 ml per pot. Conidial inoculum was produced by growing

the isolates separately on sterile oat kernels for 20 days. Spores were produced by incubating a mixture of the oat kernels infected with mycelium between two layers of plastic screen on a sand bed and placed outdoors under cool conditions (Bruehl and Machtmes, 1985).

Conidial suspensions were prepared by washing oat kernels with distilled water to harvest the spores. The suspension was filtered through layers of cheese cloth to remove mycelial and other fragments. Spores were counted with a hemacytometer and final concentration was adjusted to $1x10^5$ conidia per milliliter. Inoculated plants were incubated in a dew chamber for 48 hours. After the incubation period, plants were placed in the greenhouse at temperature of 20 °C and 15 °C during the day and night, respectively with a daylength of 9 hours.

To estimate the disease progress curve for each cultivar, pathogenicity to strawbreaker foot-rot was based on the number of leaf sheaths infected by the fungus and assessed every two weeks after inoculation. A total of six ratings were performed.

Analysis of variance and a separation of means based on Fisher's Protected LSD test were performed for each time of scoring. Estimated linear functions of infected number of leaf sheaths regressed on time (weeks) were compared using the homogeneity of regression coefficients test (Gomez and Gomez, 1984). This comparison computes a t value from two estimated regression coefficients.

Study III. Nature of Inheritance

To determine the nature of resistance to strawbreaker foot-rot in the cultivar Rendezvous , selected winter wheat crosses were evaluated. To accomplish this objective, experimental populations consisting of three parental lines and the resulting F_1, F_2 and backcross generations were obtained. The three parental lines of winter wheat were Rendezvous, Stephens, and McDermid. The desired populations were obtained by crossing the resistant Rendezvous to the two susceptible cultivars Stephens and McDermid. Observations were also made at the seedling stage to discern a possible relationship between coleoptile color and strawbreaker foot-rot resistance.

Seed of the parental lines, F_1, F_2 and backcross populations were planted 1 cm deep in plastic trays (30 cm x 22 cm x 10 cm) containing a sterilized silt loam growing medium. A randomized block design was used with 10 replications. Population sizes for the parental lines, F_1, F_2 and backcrosses included 60, 60, 400-450, and 80 seedlings, respectively. Each plastic tray contained 10 rows consisting of one row of the parental lines, F_1 and backcrosses, and 5 rows for the F_2. There were 6 seeds per row for parents and F_1, and 8-10 seeds per row for the F_2 and backcrosses. Rows were randomized within each block.

Inoculum source was produced as described in Study II. One-week-old seedlings (one leaf stage) were sprayed with a virulent isolate (PH 85-9-13 W type) of the pathogen. A conidial suspension at a concentration of 10^6, and at the rate of

10 ml per plastic tray was used. Inoculation was conducted inside a plastic chamber equipped with a misting system. Following the inoculation, seedlings were kept inside the chamber for 48 hours. They were then moved to a growth chamber for the duration of the experiment. Temperature was set at 18 °C during the day and 15 °C at night with 9 hours daylength.

Prior to inoculation, seedlings were scored for coleoptile color. Rendezvous has red (Rc3) and Stephens and McDermid have green (rc3) coleoptiles respectively, with the gene being located on chromosome 7D. Expressivity of Rc3 was not always 100 percent.

Plants were removed from the growth chamber 12 weeks after inoculation and leaf sheaths for each plant were separated and rated as previously described in Study II. The two crosses were treated as different experiments, but the procedure for both was similar.

The nature of gene action influencing disease reaction was studied by utilizing generation mean analysis. The estimation of the components of generation means was performed for each cross following the procedures describes by Singh and Chaudhary (1979).

Broad sense heritabilities estimates were computed for each cross according to the method outlined by Allard, 1960. This method consists in estimating environmental variation as the mean variance of the available nonsegregating populations. The following is the computation formula:

$H^2 = (V_P - V_E)/V_P$ where

V_P = Phenotypic variance = Variance of the F_2

V_E = Environmental variance = Mean variance of nonsegregating populations.

Narrow sense heritabilities estimates were estimated by using the backcross method as described by Fehr (1987). The following is the computation formula.

$h^2 = [2V_{F2} - (V_{B1} - V_{B2}/V_{F2})]$ where

V_{F2} is the variance among F_2 plants and V_{B1} and V_{B2} are the variances among plants from the backcross of F_1 to parent 1 and parent 2.

The number of genes controlling resistance to strawbreaker foot-rot in both crosses were estimated using the procedure described by Strausbaugh and Murray (1989).

Chi square goodness of fit tests were used to compare the actual with theoretical ratios for number of leaf sheaths infected and for coleoptile color.

RESULTS

Influence of strawbreaker foot-rot on grain yield and yield components

Favorable environmental conditions prevailed for disease development during the course of these experiments, even though lower disease severity index scores were obtained during the 1988-89 season at the Columbia Basin Research Center (CBRC). Information on the severity of strawbreaker foot-rot for 10 genotypes is presented for both seasons at CBRC and for 1988-89 at Hyslop Crop Science Laboratory (HCSL) in Figures 1, 2, and 3. Differences in disease severity were observed for treatments, genotypes and interaction between treatments and genotypes (Appendix Table 4). The interaction was mainly due to different reaction patterns among genotypes across treatments i.e. changes in ranking, crossover interactions or changes in magnitude of response.

Based on the disease severity index Genotype 1 was the most resistant, followed by Genotype 7, with all other genotypes reflecting a susceptible reaction across years and at the two locations. For the artificial inoculated plots in the 1987 growing season at CBRC (Figure 1), Genotypes 3, 4, 5, 6, 8 and 10 had disease index reactions greater than 3.5; while Genotypes 1 and 7 had scores of 1.85 and 2.75, respectively. Under the same treatment Genotype 8 had the highest disease severity index value, followed by Genotype 5. Intermediate values were noted for most

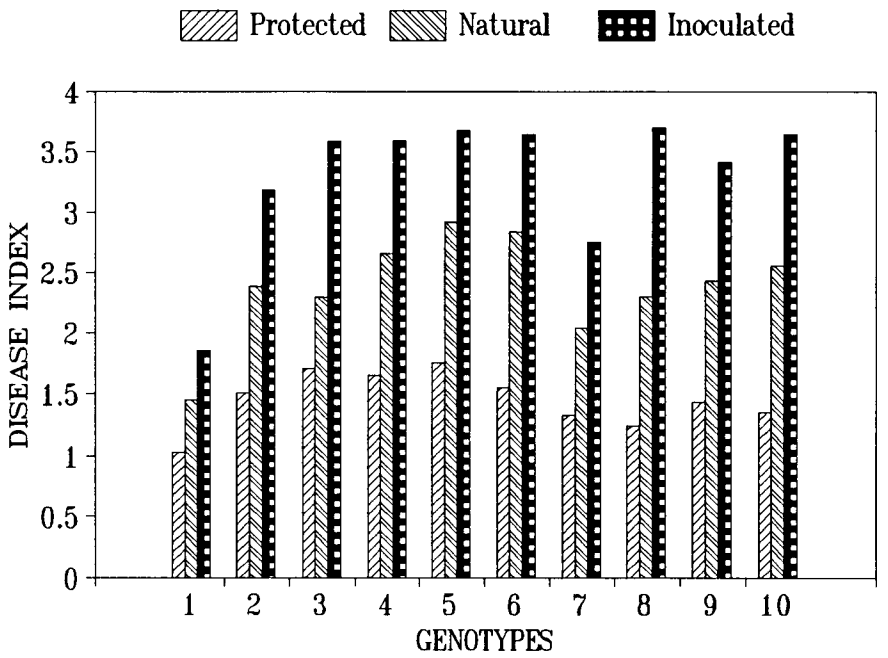

Fig. 1. Disease severity index of 10 winter wheat genotypes receiving different treatments with *Pseudocercosporella herpotrichoides* at the Columbia Basin Research Center 1987-88.
1= Rendezvous, 2= WA7423, 3= Cerco, 4= Stephens,
5= Daws, 6= McDermid, 7= Vpm/Mos 95//*2Hill,
8= Ymh/Hys//Vpm/Mos 4-2-16-17, 9= Cer/Ymh//Hys,
10= Hys/Crc, F1//Ymh/Hys

genotypes under natural infection, with Genotypes 5 and 6 having the highest disease index values. Average disease indeces for this season were 1.84, 2.4 and 3.3 for protected, natural and artificial inoculated treatments, respectively.

During the 1988-89 season at CBRC (Figure 2), the highest disease severity index scores under artificial inoculation were 3.19 and 3.07 for Genotypes 5 and 6. These two cultivars also showed a greater susceptibility under the natural inoculated treatment. Genotypes 1 and 7 again had the lowest scores across for all treatments. Larger differences in the disease severity index between natural and artificial inoculated plots were found during this growing season when compared to 1987-88. However, differences between protected and natural infected treatments were smaller. Genotypes 3 and 8 had higher scores in the protected than for the natural inoculated treatments. Mean disease severity index scores for protected, natural and artificial inoculated treatments were 0.80, 1.3 and 2.7, respectively.

When years and locations are compared, the highest disease severity index scores were recorded at the HCSL in 1988-89 (Figure 3). Genotypes 1 and 7 again had the least disease across all treatments. The most susceptible genotypes, other than 2 and 3, had scores higher than 3.5 in the artificially inoculated treatment. The difference in disease severity index between natural and artificial inoculated plots were smaller than at CBRC either in 1987-88 or 1988-89. Disease indexes of 1.3, 2.6 and 3.4 were recorded for protected, natural and artificial inoculated treatments, respectively at this location.

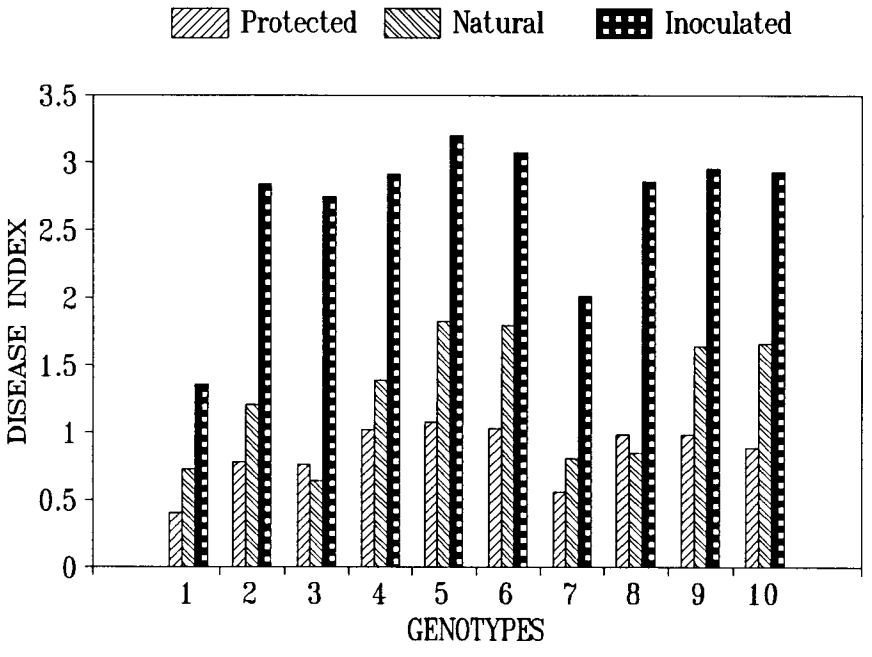

Fig. 2. Disease severity index of 10 winter wheat genotypes receiving different treatments with *Pseudocercosporella herpotrichoides* at the Columbia Basin Research Center 1988-89.
1= Rendezvous, 2= WA7423, 3= Cerco, 4= Stephens
5= Daws, 6= McDermid, 7= Vpm/Mos 95//*2Hill
8= Ymh/Hys//Vpm/Mos 4-2-16-17, 9= Cer/Ymh//Hys
10= Hys/Crc, F1//Ymh/Hys.

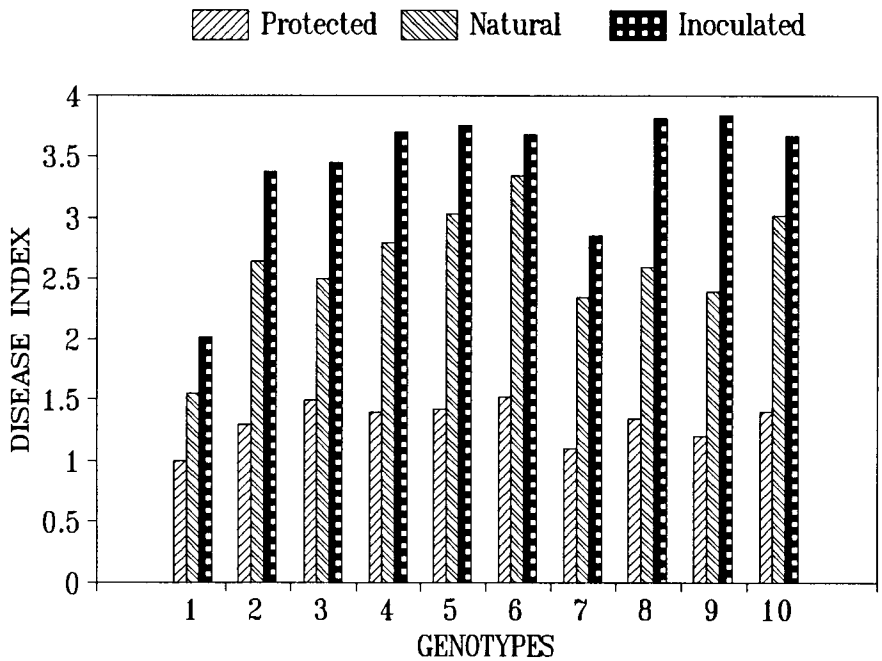

Fig. 3. Disease severity index of 10 winter wheat genotypes receiving different treatments with *Pseudocercosporella herpotrichoides* at Hyslop Crop Science Laboratory 1988-89.
1= Rendezvous, 2= WA7423, 3= Cerco, 4= Stephens
5= Daws, 6= McDermid, 7= Vpm/Mos 95//*2Hill
8= Ymh/Hys//Vpm/Mos 4-2-16-17, 9= Cer/Ymh//Hys
10= Hys/Crc, F1//Ymh/Hys

A comparison is provided between locations and treatments with reference to lodging (Table 1). It can be observed that at the CBRC site no lodging occurred among the genotypes in the protected plots in 1987-88. Lodging was apparent for Genotypes 4, 5 and 6 in the natural infected treatments. The same genotypes were severely lodged in the inoculated treatments. Only Genotypes 1 and 7 were free from any lodging. A similar response was also noted at the HCSL with again Genotypes 4, 5, 6, and also 10 being the most severely lodged. The inoculated plots again showed the most extreme lodging. Unlike the CBRC site, Genotype 7 did lodge (5%) at the HCSL. At the HCSL, Genotypes 5, 6 and 10 started to lodged shortly after heading with Genotypes 5 and 6 being lodged even in the protected plots (15 and 20% respectively). Lodging did not occur during the 1988-89 season at CBRC.

The mean yields of the 10 genotypes over years and locations for the three treatments are presented in Table 2. In the protected plots the genetic potential for grain yield did differ between genotypes at both locations with differences noted between locations and among genotypes. The largest reduction in grain yield when comparing protected, natural and inoculated treatments were observed for Genotypes 5 and 6 at the CBRC in both years. For example Genotype 5 yielded 4.9, 4.3 and 2.5 t/ha for the protected, natural, and inoculated treatments, respectively. Genotypes 1 and 7 showed essentially no change in grain yield across treatments in

Table 1. Percent lodging of 10 winter wheat genotypes receiving different inoculation treatments with *Pseudocercosporella herpotrichoides* at the Columbia Basin Research Center (CBRC) and Hyslop Crop Science Laboratory (HCSL).

	Lodging (%)					
	C B R C 1987-88			H C S L 1988-89		
Genotype[b]	P[a]	N	I	P	N	I
1	0	0	0	0	0	0
2	0	5	20	0	6	15
3	0	0	34	0	0	20
4	0	24	88	0	20	55
5	0	30	99	15	63	96
6	0	28	97	20	100	100
7	0	0	0	0	0	5
8	0	0	44	0	0	31
9	0	0	30	0	0	39
10	0	0	50	0	28	96
$LSD_{(P=0.05)}$	NS	4.67	6.64	5.4	8.54	9.81

[a] Treatments: P= Protected with fungicide, N= Natural epidemic, I= Inoculated.

[b] Genotypes: 1= Rendezvous, 2= WA7423, 3= Cerco, 4= Stephens, 5= Daws, 6= McDermid, 7= Vpm/Mos 95//*2Hill, 8= Ymh/Hys//Vpm/Mos 4-2-16-17, 9= Cer/Ymh//Hys, 10= Hys/Crc, F1//Ymh/Hys

Table 2. Mean yield (t/ha) of 10 winter wheat genotypes receiving different inoculation treatments with *Pseudocercosporella herpotrichoides* and percentage of the protected treatment at Columbia Basin Research Center (CBRC) and Hyslop Crop Science Laboratory (HCSL).

	Yield (t/ha and % P)[a]								
	C B R C 1987-88			C B R C 1988-89			H C S L 1988-89		
Genotype[c]	P[b]	N	I	P	N	I	P	N	I
1	4.9	4.8 (98)	4.8 (97)	5.2	5.1 (99)	5.1 (98)	6.9	6.8 (97)	6.5 (95)
2	5.6	5.3 (93)	4.8 (85)	6.0	5.9 (99)	5.2 (85)	5.1	4.8 (93)	4.2 (82)
3	5.8	5.6 (97)	4.6 (79)	6.1	6.1 (99)	5.2 (85)	6.3	5.8 (92)	5.3 (84)
4	5.9	5.3 (90)	3.6 (61)	5.2	5.0 (98)	4.4 (84)	7.1	5.9 (83)	4.8 (68)
5	4.9	4.3 (87)	2.5 (51)	4.8	4.6 (95)	3.7 (76)	5.9	4.5 (76)	2.8 (47)
6	5.0	4.0 (81)	2.9 (58)	4.8	4.6 (96)	3.7 (77)	5.2	2.1 (40)	1.6 (31)
7	5.6	5.5 (98)	5.2 (93)	5.5	5.3 (97)	5.3 (95)	6.5	6.0 (92)	5.9 (91)
8	5.9	5.3 (90)	4.0 (69)	6.2	6.2 (99)	5.3 (85)	6.4	5.8 (91)	4.6 (73)
9	5.9	5.7 (96)	4.4 (75)	5.6	5.5 (97)	4.7 (84)	6.6	6.1 (92)	4.9 (74)
10	5.6	4.7 (84)	3.5 (63)	5.6	5.2 (96)	4.7 (84)	5.9	5.2 (88)	3.1 (52)
$LSD_{(P=0.05)}$	0.365	(7.9)	(7.6)	0.285	(3.4)	(4.3)	0.324	(5.02)	(6.6)

[a] Percentage of protected given in parenthesis

[b] Treatments: P= Protected with fungicide, N= Natural epidemic, I= Inoculated

[c] Genotypes: 1= Rendezvous, 2= WA7423, 3= Cerco, 4= Stephens, 5= Daws, 6= McDermid, 7= Vpm/Mos 95//*2Hill, 8= Ymh/Hys//Vpm/Mos 4-2-16-17, 9= Cer/Ymh//Hys, 10= Hys/Crc, F1//Ymh/Hys

either 1987-88 or 1988-89 at the CBRC site. Other genotypes varied as to the degree of yield reduction; however in every instance the artificially inoculated treatment resulted in lower grain yield.

With the exception of Genotype 2, Genotypes in the protected plots were higher yielding at HCSL when compared to either year at the CBRC site. However, again a greater reduction at HCSL was noted with the inoculated treatment when compared to the other treatments. Both Genotypes 5 and 6 reflected the greatest reduction in grain yield, with Genotype 6 being 2.1 and 1.6 t/ha for the natural and inoculated treatments respectively, compared with 5.2 t/ha in the protected plots. Very little reduction in grain yield was observed for either Genotypes 1 and 7 across the same treatments.

Test weight was significantly or reduced in both natural and inoculated treatments at both locations with the effect of strawbreaker foot-rot being more severe in the artificial inoculated treatments (Table 3). Greater reductions in test weight were observed for Genotypes 5 and 6 at the CBRC in 1987-88. Genotypes 1, 7 and 9 showed little change across treatments during this season. During the 1988-89 season at CBRC Genotypes 6 and 9 had the lowest test weight in the inoculated plots, with the remaining genotypes showing only small reductions.

Lower test weights were noted at the HCSL, with Genotypes 4, 5, 6 and 8 showing the largest reduction in both the natural and artificial inoculated treatments. Genotypes 1, 2, 3, and 7 were not significantly reduced in test weight for any of the treatments.

Table 3. Mean test weight (Kg/m^3) of 10 winter wheat entries receiving different inoculation treatments with *Pseudocercosporella herpotrichoides* and percentage of the protected treatment at the Columbia Basin Research Center CBRC) and Hyslop Crop Science Laboratory (HCSL).

	Test Weight (Kg/m^3 and % P)[a]								
	C B R C 1987-88			C B R C 1988-89			H C S L 1988-89		
ENTRY[c]	P[b]	N	I	P	N	I	P	N	I
1	712	704 (99)	708 (99)	793	785 (99)	783 (99)	787	782 (99)	780 (98)
2	778	770 (99)	759 (97)	853	833 (98)	839 (99)	862	857 (99)	836 (97)
3	789	767 (97)	764 (97)	863	852 (98)	841 (97)	825	819 (99)	806 (97)
4	786	770 (98)	759 (97)	809	799 (99)	787 (97)	802	780 (97)	754 (94)
5	787	776 (98)	756 (96)	827	824 (99)	808 (98)	815	776 (95)	752 (92)
6	774	762 (98)	741 (96)	828	810 (98)	784 (95)	775	726 (94)	709 (91)
7	771	776 (101)	767 (99)	854	846 (99)	844 (99)	825	811 (98)	780 (97)
8	785	782 (99)	760 (97)	847	839 (99)	827 (98)	824	786 (95)	767 (93)
9	799	787 (98)	778 (99)	846	827 (98)	810 (96)	816	812 (99)	779 (95)
10	775	766 (99)	758 (98)	850	837 (98)	823 (97)	807	790 (98)	766 (95)
$LSD_{(P= 0.05)}$	7.6	(1.42)	(1.44)	10.4	(1.32)	(1.13)	7.25	(1.48)	(1.61)

[a] Percentage of protected given in parenthesis.
[b] Treatments: P= Protected with fungicide, N= Natural epidemic, I= Inoculated.
[c] Genotypes: 1= Rendezvous, 2= WA7423, 3= Cerco, 4= Stephens, 5= Daws, 6= McDermid, 7= Vpm/Mos95//*2Hill, 8= Ymh/Hys//Vpm/Mos 4-2-16-17, 9= Cer/Ymh//Hys, 10= Hys/Crc,F1//Ymh/Hys.

Mean values for 1000 kernel weight of 10 genotypes over years and locations for the three disease treatments are presented in Table 4. Kernel weight was reduced in both the naturally and artificially inoculated treatments with greater reductions observed among susceptible genotypes. In both years at the CBRC site differences in ranking of genotypes were found. However Genotypes 5 and 6 had consistently lower seed weight in the inoculated treatments. Greater reduction in kernel weight was observed at CBRC in 1987-88 in artificially inoculated plots when comparing both years. Again Genotypes 1 and 7 were less affected at this location over years.

At the HCSL site all genotypes, with the exception of Genotype 6, had higher kernel weight values in protected plots when compared to CBRC. However, greater reductions in kernel weight were noted under both inoculated treatments. Genotypes 5, 6, 9,and 10 had the greatest reduction in kernel weight, with Genotype 6 being 37.9, 27.8, and 27.9 g for the protected, natural and artificial inoculated treatment, respectively. Genotype 7 did have a significantly lower kernel weight, while no apparent reduction was observed for Genotype 1 across treatments.

Data on the number of kernel per spike for the 10 genotypes at two locations involving three treatments is presented in Table 5. The number of kernels per spike was not greatly affected at the CBRC site under natural infection, with some genotypes having more seed per spike in this treatment when compared with the protected. Fewer kernels per spike were observed in the artificial inoculated plots, with Genotypes 5, 6 and 10 having the greatest reduction. No change across

Table 4. Mean values for 1000 kernel weight (g) of 10 winter wheat entries receiving different inoculation treatments with *Pseudocercosporella herpotrichoides* and percentage of the protected treatment at Columbia Basin Research Center (CBRC) and Hyslop Crop Science Laboratory (HCSL).

	1000 Kernel weight (g) and % P)[a]								
	C B R C 1987-88			C B R C 1988-89			H C S L 1988-89		
ENTRY[c]	P[b]	N	I	P	N	I	P	N	I
1	38.5	37.4 (97)	37.5 (97)	39.1	38.4 (98)	38.7 (99)	43.2	42.3 (98)	42.2 (98)
2	36.3	35.1 (97)	33.9 (93)	33.4	32.7 (98)	30.8 (92)	42.6	40.6 (95)	35.4 (83)
3	38.3	36.7 (96)	35.2 (92)	37.6	36.3 (96)	36.9 (98)	41.4	38.9 (94)	35.8 (86)
4	43.9	41.8 (95)	39.4 (90)	34.2	33.1 (97)	31.5 (92)	46.4	43.0 (93)	39.8 (86)
5	41.5	40.1 (97)	33.9 (82)	35.7	33.5 (91)	32.6 (91)	41.7	36.4 (87)	31.7 (76)
6	38.1	35.7 (94)	33.1 (87)	34.4	32.3 (94)	31.1 (90)	37.9	27.8 (73)	27.9 (74)
7	35.3	35.0 (99)	34.4 (97)	33.0	32.6 (99)	32.3 (98)	37.2	34.5 (93)	34.0 (91)
8	33.9	33.2 (98)	31.6 (93)	36.4	35.7 (98)	35.5 (98)	39.0	35.8 (92)	34.0 (87)
9	36.4	35.9 (99)	33.7 (93)	32.7	30.8 (94)	30.2 (92)	39.2	36.1 (95)	29.6 (78)
10	36.3	33.9 (93)	33.3 (92)	32.3	30.3 (94)	30.8 (95)	39.7	35.6 (90)	30.7 (77)
$LSD_{(P=0.05)}$	1.56	(5.46)	(4.56)	1.63	(5.70)	(5.70)	1.34	(3.49)	(3.83)

[a] Percentage of protected given in parenthesis [b] Treatments: P = Protected with fungicide N = Natural epidemic I = Inoculated
[c] 1= Rendezvous, 2= WA7423, 3= Cerco, 4= Stephens, 5= Daws, 6= McDermid, 7= Vpm/Mos 95//*2Hill
8= Ymh/Hys//Vpm/Mos 4-2-16-17, 9= Cer/Ymh//Hys, 10= Hys/Crc, F1//Ymh/Hys.

Table 5. Number of kernels per spike of 10 winter wheat entries receiving different inoculation treatment with *Pseudocercosporella herpotrichoides* treatment at the Columbia Basin Research Center (CBRC) and Hyslop Crop Science Laboratory (HCSL).

	Number of kernels/spike and % P[a]					
	C B R C 1988-89			H C S L 1988-89		
ENTRY[c]	P[b]	N	I	P	N	I
1	49.0	51.0 (104)	48.7 (100)	39.7	37.6 (95)	37.6 (95)
2	61.9	62.9 (102)	59.4 (96)	46.1	44.2 (96)	42.5 (92)
3	48.9	52.0 (106)	48.2 (99)	38.1	37.2 (98)	36.7 (96)
4	59.6	59.1 (99)	57.3 (96)	38.4	37.0 (98)	35.3 (94)
5	58.1	56.1 (97)	54.4 (94)	40.0	37.1 (93)	35.9 (89)
6	54.8	53.7 (97)	52.1 (95)	35.8	33.0 (92)	29.8 (83)
7	52.2	52.9 (101)	51.9 (99)	47.8	45.6 (98)	45.4 (98)
8	51.5	52.5 (102)	50.0 (97)	44.9	41.7 (93)	40.4 (90)
9	62.2	66.0 (106)	61.2 (98)	51.0	50.2 (98)	48.5 (95)
10	52.6	51.3 (98)	50.0 (95)	40.7	35.8 (88)	36.6 (90)
$LSD_{(P=0.05)}$	3.61	(7.67)	(4.27)	4.5	(6.93)	(6.24)

[a] Percentage of protected given in parenthesis
[b] Treatments:P= Protected with fungicide, N = Natural epidemic, I = Inoculated
[c] 1= Rendezvous, 2= WA7423, 3= Cerco, 4= Stephens, 5= Daws
6= McDermid, 7= Vpm/Mos 95//*2Hill
8= Ymh/Hys//Vpm/Mos 4-2-16-17, 9= Cer/Ymh//Hys
10= Hys/Crc, F1//Ymh/Hys

treatments was observed for Genotypes 1, 7, and 9. When comparing locations, fewer kernels per spike were found among genotypes and across treatments at HCSL. Also at this location a greater reduction was observed with both the artificial inoculated naturally infected treatments. Genotypes 5, 6, 8, and 10 showed the greatest reduction in kernels per spike under the naturally infected treatment, with Genotype 10 being more affected than under the artificial inoculated treatment. Genotype 1 had the same reduction in both artificial and natural inoculated treatments. Under the artificial inoculated plots, Genotypes 5 and 6 had the greatest reduction. Only a small reduction in kernels per spike was noted for Genotype 7 when all treatments are considered.

Fewer spikes per square meter were observed in inoculated plots at the CBRC location, although Genotypes 1, 7, 8, and 10, did not showed any change in the naturally infected treatment (Table 6). When comparing the three treatments, the largest reductions in spikes per square meter were observed for Genotypes 5 and 6. Spikes per square meter for Genotypes 1 and 7 were least affected at this site, with other genotypes being intermediate in reduced values for spikes per square meter.

As is apparent in Table 6, most genotypes in the protected plots at HCSL had more spikes per square meter than were observed at CBRC locations. However, a greater reduction in spikes per square meter was observed at this

Table 6. Number of spikes/m^2 for 10 winter wheat entries receiving different inoculation treatment with *Pseudocercosporella herpotrichoides* and percentage of the protected treatment at Columbia Basin Research Center (CBRC) and Hyslop Crop Science Laboratory (HCSL).

Number of spikes/m^2 and % P[a]						
		C B R C 1988-89		H C S L 1988-89		
ENTRY[c]	P[b]	N	I	P	N	I
1	243	251 (103)	240 (98)	377	368 (97)	355 (94)
2	279	257 (92)	249 (89)	309	290 (93)	250 (81)
3	296	280 (95)	238 (80)	378	330 (87)	336 (89)
4	264	261 (99)	243 (92)	366	318 (89)	273 (76)
5	255	242 (95)	216 (84)	342	263 (77)	156 (47)
6	256	232 (91)	202 (79)	354	156 (45)	142 (40)
7	257	259 (100)	240 (93)	343	309 (90)	315 (91)
8	305	306 (100)	268 (88)	330	301 (91)	251 (76)
9	266	264 (99)	245 (92)	352	327 (93)	261 (74)
10	270	270 (100)	239 (89)	347	300 (87)	221 (64)
$LSD_{(P=0.05)}$	30	NS	(6.70)	32	(7.82)	(6.09)

[a] Percentage of protected given in parenthesis
[b] Treatments: P = Protected with fungicide N = Natural epidemic
I = Inoculated
[c] 1= Rendezvous, 2= WA7423, 3= Cerco, 4= Stephens, 5= Daws
6= McDermid, 7= Vpm/Mos 95//*2Hill
8= Ymh/Hys//Vpm/Mos 4-2-16-17, 9= Cer/Ymh//Hys
10= Hys/Crc, F1//Ymh/Hys

location with the natural and artificial inoculated treatments. All genotypes showed fewer spikes per square meter in the artificial inoculated treatment. Genotypes 4, 5, 6, 8, 9, and 10 showed the greatest reduction, with Genotype 6 having 156 and 142 spikes per square meter for the natural and inoculated treatments, respectively, as compared with 354 spikes per square meter in the protected plots. Genotypes 1 and 7 were the least affected by the inoculations in terms of spike number per unit of area.

Associations between disease severity index with grain yield, test weight and 1000 kernel weight for the 1987-88 season at CBRC are presented in Table 7. Significant negative correlations were found between grain yield and disease severity index among all genotypes, except Genotype 1. There was also a negative association between test weight and disease severity index for all genotypes, except for Genotypes 1 and 7. The association between disease severity index and 1000 kernel weight was again negative, but correlations were significant only for Genotypes 3, 4, 5, 6, 9 and 10. A similar patten was found during the 1988-89 crop season at the CBRC site (Table 8). Again no significant associations were noted for either genotypes 1 or 7 for any of the attributes measured. The largest negative associations between disease severity index and yield per plot were shown by Genotype 5 followed by Genotypes 6 and 10. For test weight the highest negative associations were found with Genotypes 6, 9 and 10. For 1000 kernel weight negative associations were computed only for Genotypes 2, 4, and 6. When kernels per spike are measured Genotypes 5, 6 and 10 reflect negative

Table 7. Coefficients of correlation between disease severity index and grain yield test weight and 1000 kernel weight for 10 winter wheat entries receiving different inoculation treatments (protected with fungicide, natural epidemic and inoculated) with *Pseudocercosporella herpotrichoides* grown at the Columbia Basin Research Center 1987-88.

Entry	Yield per plot	Test weight	1000 kernel weight
1†	0.185	0.281	-0.199
2	-0.792**	-0.583*	-0.303
3	-0.803**	-0.323**	-0.662*
4	-0.928**	-0.747**	-0.873**
5	-0.792**	-0.839**	-0.642*
6	-0.708**	-0.719**	-0.702*
7	-0.555*	-0.090	0.087
8	-0.828**	-0.579*	-0.492
9	-0.842**	-0.582*	-0.697*
10	-0.901**	-0.814**	-0.727**

Coefficients followed by ** and * are significantly different from "0" at the 0.01 and 0.05 probability levels respectively. (N = 12)

† 1= Rendezvous, 2= WA7423, 3= Cerco, 4= Stephens, 5= Daws
6= McDermid, 7= Vpm/Mos 95//*2Hill
8= Ymh/Hys//Vpm/Mos 4-2-16-17, 9= Cer/Ymh//Hys
10= Hys/Crc, F1//Ymh/Hys

Table 8. Coefficients of correlation between disease severity and yield or yield components for 10 winter wheat entries receiving different inoculation treatments (protected with fungicide, natural epidemic, and inoculated) with *Pseudocercosporella herpotrichoides* grown at the Columbia Basin Research Center 1988-89.

Entry	Yield per plot	Test weight	1000 kernel weight	Kernels per spike	Spikes per m^2
1†	0.009	-0.340	0.169	-0.142	-0.066
2	-0.732**	-0.100	-0.752**	-0.461	-0.164
3	-0.753**	-0.721**	-0.112	-0.409	-0.706*
4	-0.742**	-0.600*	-0.701*	-0.471	-0.778**
5	-0.926**	-0.795**	-0.573	-0.634*	-0.797**
6	-0.913**	-0.884**	-0.669*	-0.604*	-0.939**
7	-0.439	-0.472	-0.461	-0.217	-0.531
8	-0.734**	-0.628*	-0.229	-0.365	-0.639*
9	-0.613*	-0.879**	-0.489	-0.292	-0.524
10	-0.875**	-0.805**	-0.426	-0.685*	-0.758**

Coefficients followed by ** and * are significantly different from "0" at the 0.01 and 0.05 probability levels respectively. (N = 12)

† 1= Rendezvous, 2= WA7423, 3= Cerco, 4= Stephens, 5= Daws
6= McDermid, 7= Vpm/Mos 95//*2Hill
8= Ymh/Hys//Vpm/Mos 4-2-16-17, 9= Cer/Ymh//Hys
10= Hys/Crc, F1//Ymh/Hys

correlation values with disease severity index. These same genotypes, along with 3, 4 and 8 also had significant negative correlation values when disease severity index and spikes per square meter are considered.

In Table 9 the correlation coefficients of disease severity index and the attributes measured at HCSL are reported. Larger negative associations are revealed when compared to the data obtained at the CBRC. Significant negative association for all genotypes, except Genotype 1 were found for yield per plot, test weight and 1000 kernel weight. Kernels per spike and disease severity index were significantly correlated with the highest values noted for Genotypes 5 and 6, followed by 2, 10, and 8. When spikes per square meter are considered all genotypes including Genotype 1 resulted in significantly negative associations with the disease severity index.

Comparing locations, years and attributes measured, Genotypes 5 and 6 appeared to be most influenced by infection. Depending on the location and years, Genotypes 2, 3, and 4 were also affected by the disease. When comparing experimental sites, grain yield per plot, spikes per square meter, and 1000 kernel weight were more affected by both the natural and artificial inoculated treatments at HCSL.

Table 9. Coefficients of correlation between disease severity and yield or yield components for 10 winter wheat entries receiving different inoculation treatments (protected with fungicide, natural epidemic and inoculated) with *Pseudocercosporella herpotrichoides* grown at Hyslop Crop Science Laboratory 1988-89.

Entry	Yield per plot	Test weight	1000 kernel weight	Kernels per spike	Spikes per m^2
1†	-0.203	-0.134	-0.528	-0.267	-0.749**
2	-0.873**	-0.727**	-0.790**	-0.629*	-0.737**
3	-0.777**	-0.791**	-0.810**	-0.004	-0.739**
4	-0.875**	-0.907**	-0.939**	-0.399	-0.923**
5	-0.970**	-0.912**	-0.962**	-0.880**	-0.940**
6	-0.940**	-0.902**	-0.909**	-0.795**	-0.883**
7	-0.592*	-0.859**	-0.690*	-0.300	-0.843**
8	-0.804**	-0.926**	-0.931**	-0.589*	-0.914**
9	-0.876**	-0.853**	-0.929**	-0.389	-0.941**
10	-0.888**	-0.863**	-0.960**	-0.603*	-0.913**

Coefficients followed by ** and * are significantly different from "0" at the 0.01 and 0.05 probability levels respectively. (N = 12)

† 1= Rendezvous, 2= WA7423, 3= Cerco, 4= Stephens, 5= Daws
6= McDermid, 7= Vpm/Mos 95//*2Hill
8= Ymh/Hys//Vpm/Mos 4-2-16-17, 9= Cer/Ymh//Hys
10= Hys/Crc, F1//Ymh/Hys

Effect of inoculum concentration.

Data on the effect of spore concentration on disease severity for three winter wheat cultivars is presented in Table 10. Beginning with the first disease severity assessed 12 weeks after inoculation, there was a continued increase in disease expression as concentrations of spore suspensions increased from 10 to 10^6 spores/ml for Stephens and McDermid. No symptoms of the disease were observed at lower inoculum concentrations (10 and 100 spore/ml) for the cultivar Rendezvous. Primary disease symptoms first appeared for this cultivar at a spore concentration of 10^3 conidia/ml with the maximum disease severity observed with 3.33 leaf sheaths infected at 10^6 spores/ml. This was the same disease severity reading found for the cultivar Stephens at a lower inoculum concentration (10^3 spores/ml). Twelve weeks after inoculation, Stephens and McDermid had 2.0 and 2.67 leaf sheaths infected by the pathogen at the lowest spore concentration 10 spores/ml), respectively. At the highest spore concentration (10^6) a mean of 7.50 and 8.49 leaf sheaths completely penetrated by the fungus were recorded for the same two cultivars.

The linear regression analysis shows a positive association between disease severity and inoculum concentration. Regression equations for Rendezvous, Stephens and McDermid cultivars and their corresponding coefficients of correlations were $y = -0.779 + 0.669x$, $r = 94.3$, $y = 0.308 + 1.178x$, $r = 95$ and $y = 0.803 + 1.28x$, $r = 94.6$, respectively.

Table 10. Pathogenicity of *Pseudocercosporella herpotrichoides* as measured by the number of leaf sheats infected on seedlings of three winter wheat cultivars at different spore concentrations 12 weeks after inoculation when grown under greenhouse conditions.

Cultivar	Spore concentration (spores/ml)							Regression Slope†
	10	10^2	10^3	10^4	10^5	$5x10^5$	10^6	
Rendezvous	0	0	1.3	1.82	2.53	2.98	3.33	0.67*
Stephens	2.0	2.52	3.33	4.72	6.27	7.27	7.50	1.18
McDermid	2.67	3.17	4.17	6.33	7.67	8.33	8.49	1.28

† Value of regression coefficient b in $y = a + bx$ where y is the number of leaf sheaths infected and x is $\log_{10}$ (spore concentration).

* Significant at the 0.05 probability level, based on a t test of homogeneity of regression coefficients.

The rate of infection as measured by the slope of the regression lines of successive leaf sheaths penetrated by strawbreaker foot-rot was similar for both Stephens and McDermid. A significantly different (P = 0.05) rate of infection was found for Rendezvous (0.67) when compared to the other two cultivars based on a t test of homogeneity of regression coefficients. Lower rates of disease development were observed for Rendezvous with 0.67 leaf sheaths becoming infected for every increase in inoculum concentration from 10^3 to 10^6 spores/ml. In contrast the rate of penetration of the leaf sheaths was two-fold higher for the susceptible cultivars and 1.28 leaf sheaths being infected for every increase in spore concentration from 10 to 10^6 conidia/ml. Figure 4 shows the mean leaf sheaths infected at each concentration level for the three cultivars.

Disease progress over time.

Disease severity caused by strawbreaker foot-rot over time on seedlings of Rendezvous and McDermid is presented in Table 11. Seedlings of Rendezvous did not develop disease symptoms during the first two weeks after inoculation. The susceptible cultivar McDermid had a disease severity of 0.47, in which all the coleoptiles and part of the first leaf sheaths were infected during the same period.

As with the inoculum concentration experiment the number of leaf sheaths infected by the pathogen increased over time. However the increase was not linear. A faster increase in disease development was observed in McDermid, with significant

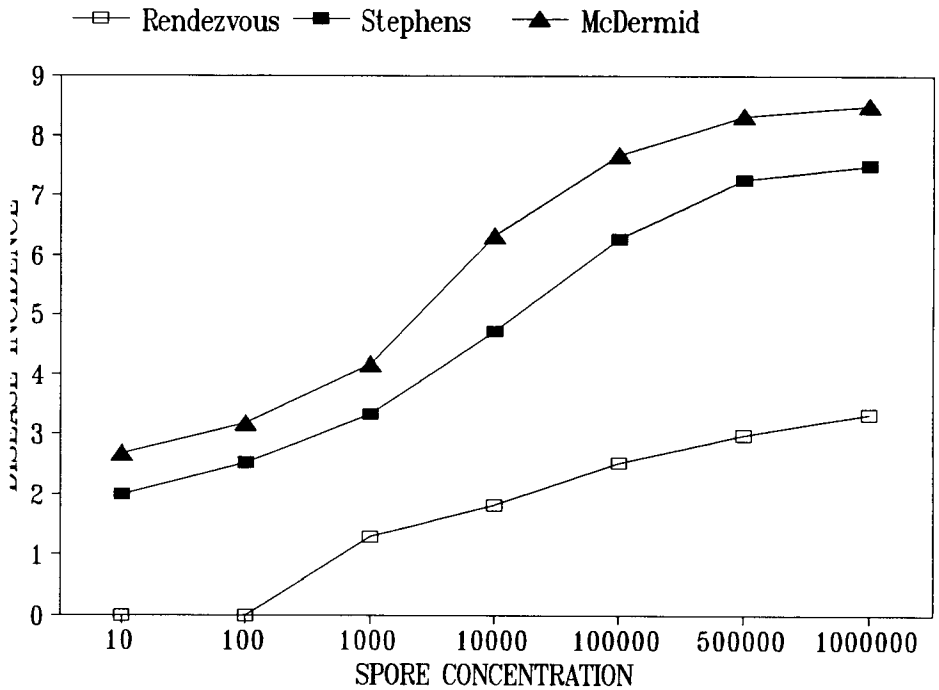

Fig. 4 Relationship between disease incidence on infected leaf sheaths and inoculum concentration for three winter wheat cultivars inoculated with *Pseudocercosporella herpotrichoides*.
Regression equations: Rendezvous, $y = 0.779 + 0.669x$, $r^2 = 94.3$; Stephens, $y = 0.308 + 1.1778x$, $r^2 = 95.1$; McDermid, $y = 0.803 + 1.28x$, $r^2 = 94.6$.

Table 11. Mean disease severity of *Pseudocercosporella herpotrichoides* as measured by the number of leaf sheats infected on seedlings of two winter wheat cultivars at different time intervals, when grown under greenhouse conditions.

	Time after inoculation (weeks)						
Cultivar	2	4	6	8	10	12	Regression Slope
Rendezvous	0.0a†	0.36a	0.58a	1.37a	1.64a	2.33a	0.231*
McDermid	0.47b	2.42b	4.22b	5.29b	6.55b	7.63b	0.704

† Means within the same column followed by different letter are significantly different. Protected least significant difference test ($P<0.05$).

* Significant at the 0.05 probability level, based on a t test of homogeneity of regression coefficients.

differences noted among the two cultivars for disease severity at each scoring period starting at two weeks. By the end of 12 weeks 7.63 leaf sheaths had been infected for McDermid compared with only 2.33 infected leaf sheaths for Rendezvous. The rate of penetration was also more pronounced in McDermid than in Rendezvous (Table 11).

The linear regression analysis performed on data collected on both cultivars revealed a positive association between disease severity and time (weeks after inoculation). Regression equations for Rendezvous and McDermid and their corresponding coefficients of correlations were $y = -0.575 + 0.231x$; $r = 0.94$, and $y = -0.506 + 0.704x$; $r = 0.97$, respectively. The rate of successive leaf sheath infection and penetration by strawbreaker foot-rot was 0.201 with an average of 0.2 leaf sheaths infected per week for Rendezvous. For McDermid the rate of successive leaf sheaths infected was 0.70 or an average of 0.7 per week. The rate of penetration among the two cultivars was significantly different based on a t test of homogeneity of regression coefficients. Results of this experiment place the cultivars in a similar ranking in terms of disease severity as previously found under field conditions. Seedling and adult plant resistance measurments are comparable.

The mean number of leaf sheaths infected at each scoring period is shown in Figure 5. The number of leaf sheaths infected by *P. herpotrichoides* increased at a higher rate in the susceptible cultivar as compared with Rendezvous. In neither case increase was there linear response.

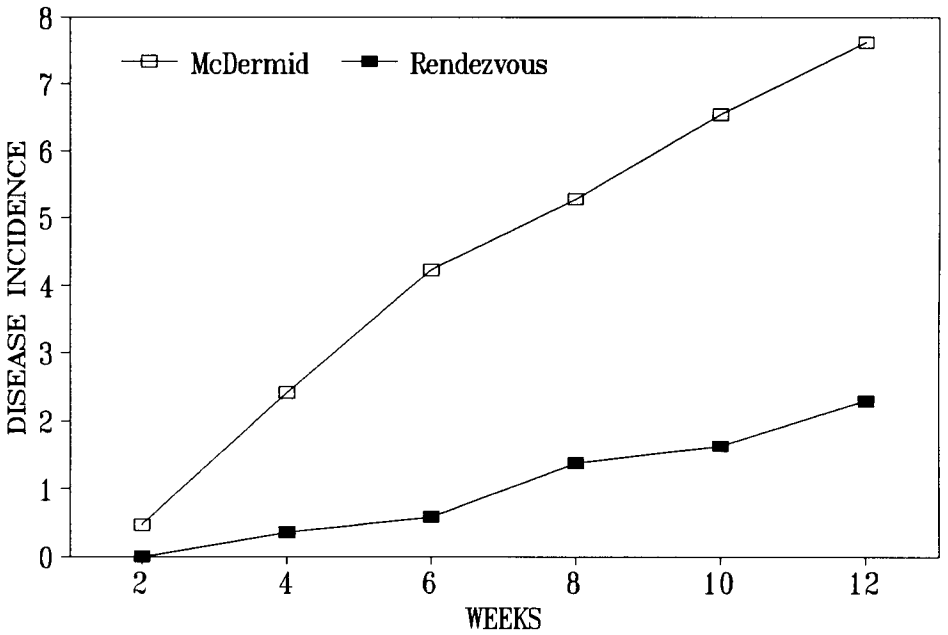

Fig. 5. Disease progress curve of *Pseudocercosporella herpotrichoides* as measured by the number of leaf sheaths infected in seedlings of two winter wheat cultivars grown under greenhouse conditions. Regression equations: Rendezvous, $y = -0.575 + 0.231x$, $r^2 = 94.0$; McDermid, $y = 0.506 + 0.704x$, $r^2 = 97.1$.

Total and percentage data on infected tillers are presented in Table 12. At week four Rendezvous had only 21.2% of its tillers infected compared with 52.8% for McDermid. Again the number of tillers infected by the fungus increased at a faster rate in the cultivar McDermid. McDermid had 96.6% of its tillers completely infected by the end of 12 weeks, in contrast to Rendezvous where less than 50% tillers were infected by the pathogen.

Inheritance and nature of gene action associated with the resistant reaction to strawbreaker foot-rot in crosses with the cultivar rendezvous.

Significant differences were observed for disease severity among generations in the crosses between Rendezvous with Mcdermid and Stephens (Table 13). The coefficients of variation were relatively high for this trait, with almost the same degree of variation noted among the two crosses (19.6 and 20.3, respectively).

The generation means and protected LSD multiple comparisons for parents and the five resulting populations are given in Table 14. In comparing generation means, the parental genotypes in both crosses exhibited the most contrasting differences. Disease severity mean values ranged from 2.82 for the resistant parent (P_2) to 8.32 leaf sheaths infected for the susceptible parent (P_1) in the cross Rendezvous x McDermid. Lower disease values for the parental lines were noted in the cross Rendezvous x Stephens with mean values of 2.71 and 7.46 leaf sheaths infected for the resistant and susceptible parents, respectively.

Table 12. Total, infected and percentage infected tillers by *Pseudocercosporella herpotrichoides* of two winter wheat cultivars at different time intervals when grown under greenhouse conditions.

	Cultivar					
	Rendezvous			McDermid		
Weeks after Inoculation	Total	Infected	% Infected	Total	Infected	% Infected
4	250	38	21.2	343	181	52.8
6	221	54	24.4	237	186	78.5
8	240	78	32.6	358	327	91.3
10	293	127	43.3	242	230	95.0
12	274	130	47.4	350	338	96.6

Table 13. Observed mean squares and coefficient of variations for disease severity (as measured by the number of leaf sheaths infected) to compare six generations of two crosses grown in a growth chamber.

S of V	DF	Rendezvous x McDermid	Rendezvous x Stephens
Block	9	0.5993	0.5325
Generation	5	43.793**	34.702**
Error	45	0.9452	0.9345
Coefficient of Variation		19.6	20.3

** Significant at the 0.01 probability level.

Table 14. Generation means for disease severity (as measured by the number of leaf sheths infected at 12 weeks after inoculation) in two crosses of winter wheat inoculated with one isolate of *Pseudocercosporella herpotricoides*.

Generation	Cross	
	Rendezvous x McDermid	Rendezvous x Stephens
P_1	8.32 a†	7.46 f
F_1	4.74 c	4.57 g
F_2	4.05 cd	4.17 g
BC_1	6.56 b	6.68 f
BC_2	3.34 ed	3.05 h
P_2	2.82 e	2.71 h

† Means in the same column having one letter in common are not significantly different based on a PLSD test.
P_1 = McDermid or Stephens
P_2 = Rendezvous

Deviations of the F_1 means from the mid-parent value favoring the resistant parent were found in both crosses. The F_1 means were higher than the F_2 means although no significant differences were found. Backcross progeny derived from crosses to the resistant parent had lower values than the F_2 means. In both cases F_2 means were lower than the backcross derived from crosses to the susceptible parental line. The F_2 means were lower than the mid parent values in all cases. Because differences were observed between populations, a generation mean analysis was performed to determine the nature of gene action by estimating the magnitude and significance of the different genetic components: additive [d]; dominance [h]; additive x additive [i]; additive x dominance [j]; and dominance x dominance [l].

The estimates of the gene effects for resistance, as measured by disease severity, for each cross are presented in Table 15. In all cases the "m" component, representing the F_2 mean, was significantly different from zero. Significant additive, dominance and additive x additive gene effects were detected in both crosses. The higher absolute estimated value for the epistatic component additive x additive [i], compared with the dominant [h] and additive [h] effects in the cross of Rendezvous x McDermid suggests that additive x additive interactions account for most of the genetic variation among generation means.

In the cross of Rendezvous x Stephens the additive effects [d] were of a higher magnitude than additive x additive [i] and dominance [h] effects. The epistatic gene

Table 15. Estimates of the additive [d], dominance [h], and epistasis [i, j, and l] genetic effects for disease severity (as measured by the number of leaf sheaths) and corresponding standard errors in six generations of two crosses of winter wheat inoculated with *Pseudocercosporella herpotrichoides*.

Estimate	Cross	
	Rendezvous x McDermid	Rendezvous x Stephens
m†	4.05* ± 0.517	4.17* ± 0.309
[d]	3.22* ± 0.874	3.63* ± 0.379
[h]	2.77* ± 1.15	2.36* ± 1.107
[i]	3.6* ± 1.14	2.78* ± 1.260
[j]	0.47 ± 0.883	1.26 ± 0.729
[l]	-2.78 ± 1.92	-2.93 ± 1.943

† m, d, h, i, j, l, are the mean, additive, dominance, additive x additive, additive x dominance, and dominance x dominance genetic effects.
* Significantly different from zero at the 0.05 probability level.

effects, additive x dominance [j] and dominance x dominance [l] were not significantly different from zero.

As noted from the magnitude of the different types and interaction effects, additive and additive x additive epistatic effects seem to be the most important components, in both crosses. The overall pattern of gene effects for both crosses was consistent for disease resistance to strawbreaker foot-rot. Broad and narrow-sense heritability estimates were computed for disease severity in both crosses using the indirect estimates of environmental variation and employing the backcross method, respectively. Broad and narrow-sense heritabilities estimates for the cross Rendezvous x Stephens were larger than those for the Rendezvous x McDermid cross (Table 16). Considering the ratio of broad-sense and narrow-sense heritability most of the variation is additive. This is in agreement with the results from the generation mean analysis.

The two methods used to estimate the number of genes are based on the assumption that there is no linkage, no epistasis, no dominance, and all loci have equal effects. Furthermore, the backcross method of estimating the number of genes assumes that all the alleles segregating for strawbreaker foot-rot resistance are in a single parental genotype of the cross. When the backcross method was considered, the estimates of number of genes were 2.5 and 2.3 for Rendezvous x McDermid and Rendezvous x Stephens crosses respectively (Table 17). When the F_2 method was used, the number of genes estimated were 2.2 and 1.2 for the same crosses.

Table 16. Broad and Narrow sense heritabilities estimates for resistance to strawbreaker foot-rot in two crosses of winter wheat.

Heritability	Cross	
	Rendezvous x McDermid	Rendezvous x Stephens
Broad	0.55	0.61
Narrow	0.48	0.53

Table 17. Estimates of the number of genes controlling resistance to strawbreaker foot-rot as measured by the number of leaf sheaths infected in two winter wheat crosses.

Method	Cross	
	Rendezvous x McDermid	Rendezvous x Stephens
Backcross	2.5	2.3
F_2	2.2	1.2
Average	2.35	1.75

For the cross Rendezvous x McDermid the F_2 population ranged from 2 to 8 leaf sheaths infected. To determine if an association exists between purple coleoptile and resistance to strawbreaker foot-rot (both traits observed in Rendezvous) an arbitrary separation between 5 (resistant) and 6 (susceptible) infected leaf sheaths was made. As a consequence the following classes were obtained: 253 resistant with purple coleoptile, 59 resistant and green, 70 susceptible and purple, and 38 susceptible and green. This ratio did not fit a 9:3:3:1 (X^2 = 12.4, P = 0.01 - 0.005). A 30.7 percent recombination value between these traits was found.

DISCUSSION

Strawbreaker foot-rot is considered the most destructive soil-borne disease of winter wheat in the states of Washington, Idaho and Oregon. Fungicide is currently applied to 70% of the winter wheat crop in eastern Washington and Northern Idaho and extensively in Oregon to control this disease.

Cultural practices and the development of cultivars with greater tillering capacity are contributing factors affecting the incidence of strawbreaker foot-rot in the Pacific Northwest. Increased use of fertilizers and early seeding for soil erosion control have initially improved yield, but promoted this pathogen and subsequent disease expression.

Strawbreaker foot-rot can reduce grain yield due to lodging as the base of the stem is weaken in the presence of the disease. Grain yield may also be reduced due to the disease even in the absence of lodging. Thus winter wheat breeders in this region must continue to breed for resistance to this disease in their research programs. In this study strawbreaker foot-rot was shown to cause major reductions in grain yield, even in potentially high yielding cultivars like Stephens. Since it is an added cost for wheat producers to spray their fields with fungicides, genetic resistance must be sought to control this pathogen. Furthermore, with continuous applications of fungicides to control strawbreaker foot-rot, an increase of resistance by certain isolates of the pathogen have been observed in Oregon. The same problem combined with a sudden shift in pathotypes was previously reported in Europe.

Currently there are no commercial cultivars with acceptable levels of resistance available for wheat growers in the Pacific Northwest.

A significant decrease in the use of fungicides can be achieved by incorporating resistance into more adapted, high yielding cultivars for this area. With resistant cultivars available, wheat growers should be able to plant wheat earlier in the fall, resulting in less disease, lower production costs, higher yield potential, and reduced soil erosion. Until recently genetic sources which provide adequate levels of resistance have not been available to wheat breeders.

The combination of resistance from *Aegilops ventricosa* with that of Cappelle-Desprez has provided good levels of resistance in the wheat cultivar Rendezvous when grown in England. However, this source of resistance had not been previously tested in the Pacific Northwest. Nor have studies to determine the number of genes involved or the nature of gene action controlling resistance or tolerance been made when this resistance source is crossed with locally adapted germplasm. If such resistance is to be successfully incorporated into adapted wheat cultivars, it is essential to have information regarding the type of inheritance controlling this trait. An additional limitation has been in obtaining a uniform and reliable screening methodology to identify resistance to this soil borne pathogen.

This investigation focused on three aspects of strawbreaker foot-rot:
1) assessment of resistance in Rendezvous and the VPM source and its capacity to prevent losses in grain yield in the presence of the pathogen genotypes found in the Pacific Northwest, 2) development of a controlled environment screening technique,

3) determination of the inheritance of resistance to the disease in crosses of Rendezvous with adapted cultivars.

Effect of Strawbreaker foot-rot on grain yield and its components

The disease severity at two locations and over two years provided an opportunity to asses the relative differential response of selected cultivars to strawbreaker foot-rot infection. Lower disease ratings observed during the 1988-89 season at the Columbia Basin Research Center (CBRC) were due to the late seeding date, as planting was delayed until the first week of October. Previous reports (Bruehl et al. 1968, Rowe and Powelson, 1973b) found that early planting in September increased the incidence of strawbreaker foot-rot as plants were larger and had more tillers during the time of maximum spore production. Herman and Wise (1985) also noted that late seeded materials were more likely to escape infection.

Disease levels obtained during the two seasons did allow for the identification of two resistant genotypes with all other genotypes showing a susceptible reaction to the pathogen. In general, there was little genotype x environment interaction for disease severity with the resistant genotypes. Susceptible genotypes did vary in their magnitude.

The lower disease severity index scores observed at CBRC in 1988-89 did not result in severe enough damage to induce lodging. However the infection level did cause some yield losses, possibly by interfering with the normal metabolism of the

wheat plant and obstructing the normal transport of nutrients and water through the stem. Based on the results of this study the HCSL, is a more appropriate site to test genetic material for resistance to strawbreaker foot-rot. Higher disease ratings were noted and more favorable environmental condition for disease development prevail at this location. Differentiation of cultivars for resistance to strawbreaker foot-rot was independent of high rainfall location (HCSL) or dryland site (CBRC). This is in contrast to the findings of Murray and Bruehl (1985) who were able to distinguish resistant from susceptible cultivars only under arid rather than humid condition. They observed that under more humid condition only small differences existed among resistance and susceptible cultivars, with the two groups varying from year to year. In some years they did not find significant differences among Cappelle-Desprez (moderately resistant) and other very susceptible cultivars. Reactions of Rendezvous and Vpm/Mos 95//*2Hill to strawbreaker foot-rot were consistently more resistant both in inoculated and naturally infected treatments across locations and years. These results confirm that Rendezvous possesses a high level of resistance to the pathotypes observed in the Pacific Northwest as well as those in England. This resistance has been reported to be due to a single dominant gene from VPM1 and through the moderate resistance of Cappelle-Desprez (Hollins, et al. 1988), with Rendezvous representing a combination of both sources of resistance.

Reaction patterns obtained in this study support the findings of Scott and Hollins (1974) in that strawbreaker foot-rot reduces the yield of winter wheat both through direct effects on the plant and indirectly due to lodging. Evidence of direct

effects is provided by the reduction in yield and yield components of inoculated plots even when lodging was slight or absent. Indirect effects in this investigation were of higher magnitude resulting from severe lodging in most susceptible cultivars in both natural and artificial inoculated treatments.

Differences among genotypes for lodging were apparent and most susceptible genotypes did lodge under severe disease pressure. The exceptions were Rendezvous and Vpm/Mos 95//*2Hill which were free from lodging under similar treatments. Genotypes reflecting greater lodging due to strawbreaker foot-rot also showed greater yield losses with even more damaging effects when lodging occurred earlier in the season. At HCSL Daws and McDermid and Hys/Crc, F1//Ymh/Hys lodged shortly after flowering and suffered major yield reductions. In general with or without lodging, strawbreaker foot-rot does reduce grain yield and the components of yield.

Reductions in grain yield among genotypes were associated with increases in disease severity scores. However, some genotypes were more affected than others in reduced grain yields. The cultivars Daws and McDermid were the most affected as revealed by the sharp yield decline observed from the control to the natural and artificial inoculate treatments. Rendezvous and Vpm/Mos 95/*2Hill were the least affected across years and locations. Only a 5% yield reduction at the HCSL site, where the disease severity index was the greatest, was observed for Rendezvous. In England a range of 3.2 to 10 percent yield loss was reported for this cultivar when severe disease occurred (Hollins, et al. 1988). The degree of yield reduction was

always closely associated with lodging. This agrees with the findings of Scott and Hollins (1978). However Murray and Bruehl (1985) found that lodging was not the primary determinant of yield reduction, although it was somehow related.

Among the traits measured in this study, test weight was the least affected by strawbreaker foot-rot. This suggests that this trait may not be useful as a selection criterion in a breeding program aimed at developing resistant cultivars.

The greatest reduction among the components of yield in the present study was for spikes per square meter followed by 1000 kernel weight and kernels per spike. Larger reductions for these traits were observed in the artificial inoculated plots compared to the natural infected plots at both locations. The largest reduction in number of spikes was observed for all genotypes in the artificial inoculated treatment at the HCSL site with the genotypes Daws and McDermid showing the greatest reduction. The reduction in the number of spikes per square meter was important in decreasing total plot yield in most genotypes in treatments showing a disease severity greater than 3.5. Decreased tiller number has been reported as a significant cause of reduced yields by strawbreaker foot-rot, (Scott and Hollins, 1974 and Murray and Bruehl 1986). Plots receiving fungicide protection retained twice as many spikes compared to the inoculated plots. This was true for the most susceptible genotypes, while little reduction was observed for Rendezvous and Vpm/Mos 95//*2Hill with any of the treatments. Based on these results breeders should be able to use both spike number and kernel weight as a criteria when screening cultivars or selecting segregating populations for resistance to strawbreaker foot-rot

under field conditions provided protected and artificial inoculated plot comparisons are available.

The degree of association between disease severity and grain yield or the components of yield provides an estimate as to how grain yield is affected by increasing disease severity levels. In most susceptible genotypes grain yield was negatively correlated with disease severity index. No correlation or a low correlation would suggest resistance or tolerance to the disease. Correlations between specific yield components and disease severity index may reveal genotypes that can compensate through a modification of the yield components. Disease severity indeces measured at HCSL generally were higher and more negatively associated with grain yield and the components of yield than similar associations at the CBRC. Although different correlation coefficients were found at both location and in different years, they were adequate in describing disease severity and yield interactions in susceptible genotypes. According to the correlation coefficients Rendezvous appeared as the most resistant followed by Vpm/Mos 95//*2Hill. Rendezvous suffered minimum grain yield loss even though highly negative correlations for spikes per square meter and disease severity were observed. Other genotypes such as Cerco and Vpm/Mos 95//*2Hill tolerated disease severity mainly by sustaining a higher number of spikes per square meter despite the presence of high disease severity levels.

The results of this study clearly suggest that breeders can screen cultivars

and segregating progenies for resistance to strawbreaker foot-rot under field conditions using artificial inoculation and scoring for disease severity. Disease severity scores obtained under field conditions were reliable from year to year and independent of the location in identifying clearly resistant genotypes. Higher disease severity scores were detected at HCSL, where more favorable environmental conditions prevail for disease development. Therefore, it is suggested that screening genotypes for resistance to strawbreaker foot-rot in the field be carried out at HCSL with planting early in September and using inoculation concentrations of 1×10^6 spores/ml to induce intensive disease pressure. Disease severity index should be measured along with lodging and spikes per square meter. Based on these results it is also apparent that the genetic resistance found in Rendezvous and in the VPM source are effective to the pathotypes employed in this investigation.

Effect of inoculum concentration and disease development over time

Cultivars and progenies resistant to strawbreaker foot-rot can also be identified under controlled environment conditions. Under the conditions imposed in this experiment it was also possible to distinguish resistant from susceptible genotypes at each spore concentration used. The greatest difference between Rendezvous and susceptible genotypes was at 10^5 spores/ml. Furthermore, Rendezvous did not show any symptoms of the disease at 100 spores/ml when infection was observed on susceptible genotypes. As spore concentration increased,

the resistance of Rendezvous appears to be overcome by the pathogen, but still at the highest inoculum concentration Rendezvous shows a clear advantage over Stephens and McDermid.

Disease symptoms developed at a lower rate over time on Rendezvous, with no disease symptoms appearing during the first two weeks after inoculation. In contrast McDermid where 0.47 leaf sheaths were infected. Screening for resistance under greenhouse conditions can be achieved by measuring disease severity at 2 to 12 weeks after inoculation.

This screening technique can facilitate resistance breeding by providing the consistent and uniform level of infection required for discrimination among parental genotypes and their segregating progeny. As shown in the previous section of this thesis, Rendezvous and VPM provide acceptable and detectable levels of resistance under field conditions. The consistency of the disease severity index for the resistant cultivars over years and across locations indicates that the techniques employed were satisfactory in identifying resistant genotypes. In addition it would appear for the three cultivars tested, there was good agreement between adult plant disease severity readings obtained in the field and those noted in the greenhouse on seedling. Identifying resistant genotypes at the seedling stage would offer many advantages to the plant breeder. It would save time in evaluating segregating populations for disease resistance, plus the fact that seedlings found to be resistant could be then transplanted in the field for evaluation of other selection criteria.

The technique used in this study has the precision necessary to detect differences for the level of resistance found in Rendezvous. However, it may lack the precision required to distinguish small differences in resistance among genotypes encountered in segregating populations, where a high degree of replication might be needed either in the greenhouse or field conditions. This high degree of replication has been necessary to be able to detect differences between "Cappelle-Desprez" (moderately resistance) and other susceptible cultivars (Scott 1971, Koebner and Martin 1990).

Inheritance and mode of gene action

There are clearly genetic differences in strawbreaker foot-rot. In the generation mean analysis, the F_1 and F_2 means of crosses between Stephens and McDermid with Rendezvous were near the midparent values, although slightly skewed toward the resistant parent Rendezvous. Depending on the recurrent parent, the backcrosses deviated significantly toward either the resistant or susceptible recurrent parent. When estimating the nature of gene action, significant additive, dominance and additive x additive effects were observed.

The magnitude of the additive and additive x additive gene action was higher than the dominance effects. This suggests that additive and additive x additive epistatic effects played a greater role than the non-additive genetic variance in the expression of strawbreaker foot-rot resistance. Previous studies (Strausbaugh and

Murray, 1989; Saragoussi, 1986) involving VPM and Cappelle-Desprez have also shown that additive, dominance and epistatic gene effects were of major importance in controlling resistance to strawbreaker foot-rot.

Further evidence of the additive effects is provided by the narrow and broad sense heritability estimates. The large narrow sense estimates indicated that there is considerable additive genetic variance for resistance to strawbreaker foot-rot. From the relative values noted in the generation mean analysis, the estimates of gene action, and heritability estimates would suggest that the resistance found in Rendezvous is conditioned by a relatively few genes. When the number of genes were estimated it would appear that approximately two genes were involved. At least two genes would be expected if the VPM and Cappelle-Desprez sources of resistance are different as previously reported.

In a conventional breeding program, wheat breeders are concerned with genes which behave in an additive manner as this is the only type of gene action that is useable in self pollinating crops. Much of the genetic variance associated with resistance to strawbreaker foot-rot in the crosses studied was of the additive type. Based on the disease assessment study the VPM source alone would also appear promising, but does not provide the same degree of resistance as Rendezvous.

Due to the additive genetic nature of resistance and the few number of genes involved, selection in these crosses could start as early as in the F_2 generation. Additional screening would be necessary in the F_3 to verify the resistance. Screening for resistance in segregating populations of crosses involving Rendezvous as a source

of resistance can be performed at the seedling stage. Inoculation of seedlings together with resistant and susceptible checks can be carried out in the greenhouse. A range of inoculum concentration at the 2 leaf stage can be used. Plants can be scored for the disease at 12 weeks after inoculation. With 100 spores/ml, no symptoms of the disease would be noticed for genotypes possessing the Rendezvous level of resistance. Another alternative is to use a higher inoculum concentration and score for the disease severity earlier i.e. two weeks after inoculation. Again the resistant genotypes similar to Rendezvous should be free of disease at this period of time. Resistant genotypes can then be transplanted in the field, advanced, and evaluated for other traits. Screening for resistance at the seedling stage can be performed any time of the year because spores can be produced in the laboratory, thus overcoming the time consuming, environmentally sensitive method proposed by (Bruehl and Machtmes, 1985).

Breeding strategies such as natural and mass selection had been employed to breed for resistance to strawbreaker foot-rot. However these breeding methods have proven to be ineffective, especially in early generations (Roberts and Allan, 1990). Single seed descent has also been proposed as a possible breeding methodology to increase the resistance to strawbreaker foot-rot (Roberts and Allan, 1990; Strausbaugh and Murray, 1989). Contrary to their proposed methodology, these results show that wheat breeders should be able to select for increased resistance to strawbreaker foot-rot in early segregating generations and use any of a number of different breeding methods in handling advanced generations.

The question of using coleoptile color as a phenotypic marker to follow alleles conferring resistance to strawbreaker foot-rot appears possible. With the crosses employed in this study an independent assortment value of 37.5% would be expected without linkage. An association between coleoptile color and resistance was found to be 30.7% in the cross between Rendezvous (purple coleoptile) and McDermid (green coleoptile)suggesting some association, but not at a level for effective selection using coleoptile color as a marker for disease resistance. Independent segregation was also reported by Worland et al. 1988 between coleoptile color and the major gene conferring resistance in VPM1.

Recently a linkage has been demonstrated between the resistance gene located on chromosome 7D of VPM and an allele coding for endopeptidase 1 (Ep-D1b) (McMillin et al. 1986). Both genes were inherited by VPM from *Aegilops ventricosa* (Worland et al. 1988). These workers reported a very tight linkage between the two genes and suggested that the Ep-D1b biochemical marker can have a considerable advantages over traditional screening methods for strawbreaker foot-rot resistance. However, this marker is useful only for the VPM resistance gene, which is less effective than that observed in Rendezvous, where the Cappelle-Desprez resistance gene is also present. Since differences can be easily distinguished at the seedling stage among genotypes carrying the resistance of Rendezvous it is not necessary to employ the biochemical marker.

SUMMARY AND CONCLUSIONS

This investigation was conducted to determine if strawbreaker foot-rot resistance reported in the cultivars Rendezvous and VPM was effective on isolates found in the Pacific Northwest winter wheat growing areas. Information was also obtained on the use of different inoculum concentrations to assess resistance in cultivars at the seedling stage. Finally the inheritance and mode of gene action of resistance to strawbreaker foot-rot was studied in the cultivar Rendezvous in crosses with two susceptible, adapted cultivars.

These studies were designed to provide wheat breeders with a better understanding of this pathosystem and to develop strategies for identifying and incorporating resistance to strawbreaker foot-rot in high yielding cultivars.

Selected wheat cultivars with different reaction patterns were evaluated for response to strawbreaker foot-rot in field and greenhouse experiments. Under field conditions 10 genotypes were evaluated at two locations. For one location, two years of data were collected. Both natural infection and artificial inoculations were used to maximize disease expression. The effect of strawbreaker foot-rot on yield and the components of yield was estimated in this study. In the greenhouse, three cultivars were used to determine the effect of inoculum concentration on the expression of resistance at the seedling stage. The inheritance of resistance to strawbreaker foot-rot in Rendezvous was also evaluated using generation means analysis. The

experimental material consisted of parental lines, F_1, F_2 and backcrosses generations. Parental lines were chosen based on their extremes of resistance and susceptibility.

The following conclusions were reached.

1 Based on the disease severity index, Rendezvous was the most resistant genotype to strawbreaker foot-rot, followed by Vpm/Mos 95//*2hill. All other genotypes had a susceptible reaction across years and at the two locations. These results confirm that Rendezvous posseses a high level of resistance to the pathogen isolates observed in the Pacific Northwest.

2 When years and locations are compared, the highest disease severity index scores where recorded at the HCSL in 1988-89, where more favorable environmental conditions prevail for disease development.

3 Differences among genotypes for lodging were apparent and most susceptible genotypes did lodge under severe disease pressure. However Rendezvous and Vpm/Mos 95//*2Hill were free from lodging under the same treatments. Genotypes reflecting greater lodging due to strawbreaker foot-rot also showed a greater yield loss, especially when lodging occurred early in the season.

4 Higher grain yields were observed at HCSl in the protected plots when compared to either year at CBRC site. A greater reduction in grain yield was also noted in inoculated plot at the HCSL location. Very little reduction was

observed for Rendezvous and Vpm/Mos 95//*2Hill in any treatment. The degree of yield reduction was closely associated with lodging.

5 The greatest loss among the components of yield was noted for spikes per square meter, followed by 1000 kernel weight and kernels per spike. Larger reductions were observed in the artificially inoculated plots. Test weight was the least affected. The reduction in the number of spikes per square meter was important in decreasing total plot yield in most genotypes showing a disease severity grater than 3.5. Little reduction was noted for Rendezvous and Vpm/Mos 95//*2Hill.

6 It was possible to distinguish resistant from susceptible genotypes at each spore concentration used. Rendezvous did not show any symptoms of the disease at 100 spores/ml when infection was observed on the susceptible genotypes. As spore concentration increased, the resistance of Rendezvous was overcome by the pathogen, but still at the highest inoculum concentration this cultivar shows a clear advantage over susceptible cultivars. The rate of leaf sheath infection was also lower in Rendezvous compare with the other cultivars.

7 In contrast to the susceptible genotypes disease symtoms developed at a lower rate over time on Rendezvous, with no disease being noted during the first two weeks after inoculation. Disease severity measured at 12 weeks after inoculation revealed that the rate of penetration was also more pronounced in the susceptible genotype than in Rendezvous. Screening for resistance

under greenhouse conditions can be achieved by measuring disease severity at 2 to 12 weeks after inoculation, using a high inoculum concentration.

8 Generation means analysis indicated that additive, dominance and additive x additive gene action were involved in the expression of disease resistance. However, the magnitude of the additive and additive x additive gene action was higher than the dominance effects, suggesting that the additive and additive x additive genetic variance played a greater role than the non-additive genetic variance in the expression of resistance to strawbreaker foot-rot in crosses with Rendezvous.

9 Estimates of the number of genes for resistance to strawbreaker foot-rot in both crosses indicated that approximately two genes were involved. Narrow sense heritability estimates were reasonably high. Selection for resistance should be possible, and effective, in early generation segregating populations.

10 An association between coleoptile color and resistance to strawbreaker foot-rot was found, but not at a level that would be useful for indirect selection.

11 Results from this study suggest that the resistance found in Rendezvous can be selected for at the seedling stage in the greenhouse, or in field environments such as the HCSL. For seedling screening, the rate of leaf sheath penetration provided a measure of resistance. In the field, lodging, tillers per square meter, 1000 kernel weight and kernels per spike would all be effective indirect selection criteria for resistance.

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APPENDIX

Appendix Table 1. Pedigree and Description of the winter wheat entries included in these studies.

Entry 1: Cultivar Rendezvous (VPM 1/Hobit//Virtue). Soft red winter wheat developed by the Plant Breeding Institute in England. High tillering, late maturity and highly resistant to strawbreaker foot-rot.

Entry 2: WA 7433. Soft white winter wheat line developed by Washington State Agricultural Research Center. High yielding, moderately winter hardiness, moderately resistant to strawbreaker foot-rot.

Entry 3: Cultivar Cerco (Maris Huntsman/VH 74521). Semidwarf soft red winter wheat, developed cooperatively by the ARS-USDA and the Washington State Agricultural Research Center. Moderately resistant to strawbreaker foot-rot.

Entry 4: Cultivar Stephens (Nord Deprez/Pullman 101). Soft white winter wheat cultivar released by the Oregon Agricultural Experimental Station. Medium to high tillering, moderate head fertility, and susceptible to strawbreaker foot- rot.

Entry 5: Cultivar Daws (CI 14484//CI 13645/CI 178383). Soft White winter wheat developed by the ARS-USDA, and Washington State Agricultural Research Center. Susceptible to Strawbreaker foot-rot.

Entry 6: Cultivar McDermid (Nord Deprez/Pullman Sel 101). Soft winter wheat developed by the Oregon Agric. Experimental Station in cooperation with ARS-USDA. Early maturity, medium height, and susceptible to strawbreaker foot-rot.

Entry 7: (VPM/Mos 95//*2HILL). Soft white winter wheat advanced line from the Winter Wheat Breeding Program at Oregon State University. Moderately resistant to strawbreaker foot-rot.

Entry 8: (Ymh//Hys//Vpm/Mos 4-2-16-17). Soft white winter wheat advanced line from the Winter Wheat Breeding Program at Oregon State University. Susceptible to strawbreaker foot-rot.

Entry 9: (Cerco/Ymh//Hys). Soft white winter wheat advanced line from the Winter Wheat Breeding Program at Oregon State University. Susceptible to strawbreaker foot-rot.

Entry 10: (Hys/Cerco,F_1//Ymh/Hys). Soft white winter wheat advanced line from the Winter Wheat Breeding Program at Oregon State University. Susceptible to strawbreaker foot-rot.

Appendix table 2. Summary of weather data on a per month basis at the Columbia Basin Research Center, 1987-88 and 1988-89 growing season.

Growing season	Month	Precipitation (mm)	Temperature oC		
			Average Max	Average Min	Mean
1987/1988	October	0.0	22.0	-1.4	10.3
	November	36.6	11.1	-0.2	5.5
	December	40.9	4.9	-3.9	0.5
	January	66.0	4.2	-4.2	0.0
	February	8.1	10.2	-3.4	3.4
	March	41.9	13.1	-0.6	6.2
	April	65.8	17.7	3.9	10.8
	May	45.5	20.7	5.6	13.1
	June	23.9	24.8	9.2	17.0
	July	0.0	32.0	10.5	21.2
	Total	328.7			
1988/1989	October	2.0	23.1	3.7	13.4
	November	92.7	11.1	1.8	6.4
	December	27.9	5.1	-3.1	1.0
	January	72.6	7.1	-1.9	2.6
	February	39.4	0.7	-9.6	-4.4
	March	74.9	11.1	1.0	6.1
	April	49.3	18.0	3.9	11.0
	May	55.6	20.5	5.4	12.9
	June	3.8	31.1	9.7	20.5
	July	30.2	28.2	11.0	19.6
	Total	456.8			

Appendix table 3. Summary of weather data on a per month basis for the Hyslop Crop Science Laboratory, 1988-89 growing season.

Growing season	Month	Precipitation (mm)	Temperature oC		
			Average Max	Average Min	Mean
1988/1989	October	3.6	20.1	8.1	14.1
	November	276.1	11.4	4.8	8.1
	December	100.8	7.7	1.2	4.5
	January	106.2	8.3	1.6	4.9
	February	81.5	5.8	-2.2	1.8
	March	172.7	11.7	3.4	7.6
	April	36.1	18.8	6.6	12.7
	May	37.1	19.3	6.7	13.0
	June	28.9	24.5	10.3	17.4
	July	8.4	24.8	10.9	17.8
	Total	851.4			

Appendix Table 4. Observed mean squares and coefficients of variation for disease severity of 10 winter wheat entries receiving different inoculation treatments (protected with fungicides, natural, and artificial inoculated) with *Pseudocercosporella herpotrichoides* grown at the Columbia Basin Research Center (CBRC) and at Hyslop Crop Science Laboratory (HCSL).

S of V	D.F.	CBRC 1987-88	CBRC 1988-89	HCSL 1988-89
BLOCK	3	0.2918	0.5047*	0.1059
TMTS	2	9.0443**	32.719**	24.459**
ENTRIES	9	1.5742**	3.6076**	3.0001**
TMTS X ENTRIES	18	0.3696*	0.4072**	0.2277**
ERROR	87	0.1943	0.1817	0.0545
COEFFICIENT OF VARIATION		16.10	25.71	8.65

* Significant at the 0.05 probability level
** Significant at the 0.01 probability level